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Appendix IV-E

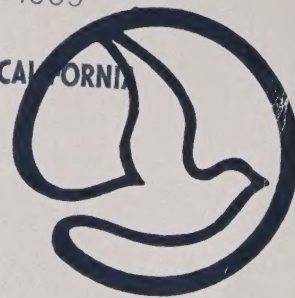
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APPENDIX IV—E
METHANOL FUEL**

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METHANOL FUEL**



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PREFACE

In order to attain state and federal ambient air quality standards within the South Coast Air Basin, the use of alternative fuels, especially methanol, is being considered. Methanol has the potential to become the primary fuel of the future, replacing conventional petroleum based fuels in the transportation as well as stationary source sectors.

Methanol plays a role in achieving the Air Quality Management Plan (AQMP) objectives. Although methanol is included in Tier I of the AQMP as an alternative to other control technologies, Tiers II and III include methanol penetration in both transportation and stationary sources.

This appendix has been prepared mainly in support of the AQMP and consists of reprints of several publications to provide some insight on the use of methanol and is further divided into four sections: Section I presents some of the recent demonstration projects performed in stationary sources (gas turbines and utility boilers) along with new developments for methanol fueled gas turbines. Section II describes two ongoing methanol programs in mobile sources and a comparative study of heavy-duty engine operation with diesel fuel and methanol. Section III deals with other safety, environmental, and economically related impacts of utilizing methanol as a primary commercial fuel. Section IV is a bibliography on various methanol related topics including technology development and evaluation, light/heavy-duty vehicle emissions data and test programs, health/environmental effects, production, distribution, and economics.

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SECTION I
METHANOL USE IN STATIONARY SOURCES

METHANOL

Clean Coal

Stationary Engine

Demonstration Project

EXECUTIVE SUMMARY

FEBRUARY 1986



**CALIFORNIA
ENERGY
COMMISSION**

P500-86-004

CONSULTANTS REPORT

CONSULTANTS
REPORT

METHANOL

Clean Coal
Stationary Engine
Demonstration Project

EXECUTIVE SUMMARY
FEBRUARY 1986

**CALIFORNIA
ENERGY
COMMISSION**

Prepared By:

**KVB, Inc.
Irvine, California**

ABSTRACT

A field test demonstration of the use of methanol in an industrial-sized gas turbine cogeneration unit was conducted in 1984 at the Central Heating Plant of the University of California at Davis. This program, sponsored by the California Energy Commission, was one of four full-scale demonstrations of the use of clean fuels in mobile and stationary engine applications. These demonstration programs were directed toward the evaluation of clean fuels potentially derived from coal, for meeting California's future energy needs.

A 3,250 kW Allison 501-KB gas turbine, designed originally for either natural gas or distillate fuel oil operation, was converted to methanol and operated for a total of 1036 hours. A methanol storage and handling facility for fueling the engine was designed and built on-site. Engine modifications specific to the utilization of methanol and preliminary test work of the specialized components were performed by the turbine manufacturer. The components were then installed on the engine and the unit was operated on methanol. Emissions and performance data were monitored throughout the test. The relatively low NO_x emissions expected from methanol operation were further reduced by the implementation of water injection via mixing with the methanol prior to supply to the engine. Engine operating problems attributable to methanol were not encountered during the demonstration testing, therefore engine performance was judged satisfactory. However difficulties of methanol pumping compatibility with the engine-driven, high-pressure fuel pump necessitated the use of an off-board centrifugal pump to complete the program.

Note: The use of company tradenames in this document does not constitute an endorsement of their products or services by the authors or the California Energy Commission.

ACKNOWLEDGEMENTS

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Special thanks is given to Jerry Keen and Ray Bailey of the Department of Physical Plant at the University of California at Davis for their unlimited patience and perseverance during the project. The project could not have been carried out without the full cooperation of these individuals and the staff of the campus Central Heating and Cooling Plant.

Thanks is given to the following individuals for their valuable assistance in performing the project:

- o Ray Lewis, Jim Snyder, Bill Adams, Ron Larsen, Don Starr, and Jim Wilson of the Celanese Chemical Company, Inc.
- o Ralph Kidder, Pompeii Hawkins, Eldon Foster, and John Latcovich of Allison Gas Turbine Operations
- o Dave Ingals, Harold Hamilton, and Diana Inslee of Williams and Lane, Inc.
- o Bill Francis of Monterey Mechanical Company

The California Energy Commission and KVB acknowledge and thank all those individuals whose participation on the team made the project possible.

THE CLEAN COAL STATIONARY ENGINE DEMONSTRATION PROJECT

REPORT VOLUME I EXECUTIVE SUMMARY

PREPARED FOR:

CALIFORNIA ENERGY COMMISSION
SACRAMENTO, CALIFORNIA

CONTRACT NO.
500-82-003

PREPARED BY:

G. H. SHIOMOTO
D. E. SHORE

KVB, INC.
ENGINEERING AND RESEARCH DIVISION

MAY 1985

KVB72-804830-1998

INTRODUCTION

The California Legislature Senate Bill 771 (Alquist, 1979) and the Budget Act of 1979 require the California Energy Commission (CEC) to develop and implement a program to evaluate the use of clean fuels derived from coal. Methanol is considered to be a very clean fuel, from the standpoint of air pollution, and can be derived from coal as well as natural gas and biomass. Methanol has been shown to be a superior fuel as it has been used for years for high performance racing automobiles.

OBJECTIVES

The CEC Clean Coal Stationary Engine Demonstration Project was performed to show that methanol is technically viable as an industrial gas turbine fuel. An existing gas turbine cogeneration facility was converted to methanol fuel with the objective of operating the facility for 1,050 hours on methanol. A principal objective of the project was to demonstrate the clean burning, low air pollution characteristics of methanol use in a gas turbine. A NO_x emission limit of 0.1 μg per Joule energy output was established as the emission goal for the project. This limit, equivalent to approximately 40 ppm (corrected to 15 percent O_2) at full plant output, represented a control measure which had been suggested in 1978 by the California Air Resources Board for cogeneration facilities. The project included the provision of a combustion system designed to minimize NO_x emissions. Another important objective was to demonstrate that the utilization of methanol as fuel for an industrial plant can indeed be accomplished safely, reliably and economically using existing engineering practices and currently available materials. An additional objective was to assemble the necessary cost information and perform an economic analysis of fueling a gas turbine cogeneration plant with methanol.

The demonstration project was conducted by a team assembled by KVB, Inc. of Irvine, California. KVB served as the prime technical and administrative contractor. The demonstration was performed at an existing gas turbine

cogeneration plant at the University of California at Davis (UCD). The host site facility includes an Allison 501-KB industrial gas turbine originally set up to operate on conventional fuels. Engine modifications necessary for methanol operation were provided by the Allison Gas Turbine Operations of the General Motors Corporation. Methanol fuel for the demonstration was supplied by the Celanese Chemical Company, Inc.

CONCLUSIONS AND RECOMMENDATIONS

The UCD Allison gas turbine was converted to methanol firing and was operated for a total of 1,036 hours. Operation on methanol began March 16, 1984 and was completed November 16, 1984. This period of time was required to complete the demonstration as a result of activities necessary to resolve unanticipated equipment problems. Initially, it was not possible to operate the engine continuously on methanol due to problems encountered in the delivery of methanol, under high pressure, to the combustion section of the engine. It was intended originally to utilize an engine-driven, gear-type pump for the high-pressure delivery of methanol. Attempts to use the engine-driven pump were unsuccessful in that the internal components of the pump failed as a result of the low lubricating qualities of methanol. A total of 126 hours of operation on methanol were accumulated with the engine-driven pump design.

In order to meet the project goals, it was necessary to implement a major modification to the project work scope. A high-pressure centrifugal pump system was selected as the replacement to the gear pump. A significant effort was necessary to engineer, design, and fabricate the centrifugal pump system in order to match the pump characteristics to those of the remainder of the methanol fuel system. Once the system was completed, 910 hours of near-continuous operation on methanol were logged. Operation of the centrifugal pump system was satisfactory during the remainder of the demonstration run.

The low emissions characteristics of a methanol-fueled gas turbine cogeneration facility were verified during the demonstration. The project objective of achieving NO_x emissions less $0.1 \mu\text{g}/\text{Joule}$ of energy output (approximately 40 ppm, corrected to 15 percent O_2 , at full plant output) was met during all periods of operation on methanol. In fact, it was possible to

meet the NO_x emissions target even without the use of water injection. Water injection was implemented simply by blending water and methanol upstream of the engine. With water injection, NO_x emission levels significantly below the project goal were realized. At full and near-full plant output, NO_x emissions in the range of 10 to 15 ppm were measured.

The long-term operation of the engine was relatively uneventful with respect to the need for extraordinary operating and maintenance activities attributable to the use of methanol. Minor problems resulted from rust in the methanol storage tank and the subsequent plugging of fuel filters. Higher-than-expected pressure losses within the fuel system dictated close attention by operating personnel. These minor problems are considered to be inconveniences attributable to the nature of the fuel system hardware utilized in the demonstration, rather than a significant flaw in the technology.

At the conclusion of the demonstration run, a teardown inspection of the engine revealed that damage had been incurred to the power section of the turbine. The damage, according to the engine manufacturer, was the result of high temperatures occurring during engine startup. The type of damage is generally considered to be a turbine control system problem, rather than related to the combustion characteristics of the fuel being used and was typical of overtemp starts which can occur on conventional fuels such as natural gas or distillate fuel oil. The engine had been operated on gas prior to the methanol run and intermittently on gas during the methanol run. Therefore, it was not possible to determine which fuel was in use when the damage occurred. Of most significance is that the type of damage was not atypical and, therefore, is not considered as a problem inherent to the use of methanol as gas turbine fuel.

The demonstration project covered a very broad range of engineering, testing, and analysis activities. Based upon the results of these activities, the following specific conclusions can be drawn:

1. The technical viability of the use of methanol as gas turbine fuel was demonstrated as a result of 1,036 hours of operation of the UCD engine.
2. The use of methanol as gas turbine fuel results in very low emissions of air pollutants. NO_x emissions were within the

project goal and were 60 percent below emission levels for natural gas firing. NO_x emissions as low as 10 ppm were demonstrated with high rates of water injection.

3. Emissions of carbon monoxide and aldehydes for high load operation on methanol were low and similar to levels measured for natural gas operation. At part load, emissions of CO and aldehydes for methanol operation increased and were higher than levels for natural gas. Optimization of fuel nozzles and associated combustion hardware is recommended in order to reduce CO and aldehyde emissions.
4. The premixing of methanol and water prior to introduction into the combustion section of the turbine is a viable and recommended method of NO_x control. The method eliminates the need for a separate high-pressure system for the injection of water directly into the combustion chamber.
5. The storage and handling system for fueling a gas turbine plant with methanol can be fabricated using conventional engineering practices and commercially available hardware and materials. Additional research in this area is neither required nor recommended.
6. Without major modifications, the use of a gear-type pump designed originally for petroleum-based liquid fuels appears to be inadequate for the high-pressure delivery of methanol in a gas turbine system. The use of a high-pressure centrifugal pump appears to be a satisfactory alternative and is the recommended pumping approach considering the current state-of-the-art. Development of a methanol-compatible engine-driven gear-type pump is also recommended since such system may be more efficient and less cumbersome than a centrifugal pump.
7. The formation and accumulation of rust inside a methanol storage tank constructed from carbon steel can be troublesome with respect to fuel filtering systems. Adequate storage tank cleaning activities and the proper design of fuel filtration systems is mandatory.
8. Given a properly designed fuel system, the operating and maintenance requirements of a methanol-fueled gas turbine facility are no different than those of a conventional-fueled plant.
9. Damage to the UCD gas turbine power section was incurred at some time before or during the demonstration on methanol. The damage could have happened during the operation on either distillate fuel oil, natural gas, or methanol and is, therefore, not necessarily attributable to the use of methanol. The damage was most likely caused by an overtemp

condition during startup of the turbine. Precaution in the design, set-up, and operation of the turbine control system is the recommended approach for preventing such problems.

10. The heat rate performance of the demonstration engine operating on methanol was equivalent to or slightly better than the performance on natural gas.
11. The cost of converting the UCD gas turbine engine to methanol operation was \$670,000. This cost is not entirely representative of the costs which would be expected to be incurred for the retrofit of a similarly-sized commercial application since the demonstration retrofit included elements which were developmental in nature.
12. The cost of converting a similarly-sized gas turbine facility to methanol operation has been estimated to be within a relatively wide range of \$420,000 to \$769,000. The variability is due to the several elements of a given retrofit project that would be site specific and the fact that engine components required for a methanol conversion are currently not commercially available.
13. For a new gas turbine installation, the cost difference between a methanol-fueled and a conventional-fueled system is estimated to be negligible to as much as \$497,000 (\$155/kW) more for a methanol system. This is in comparison to a cost of \$1,000/kW to \$1,500/kW for an entire cogeneration facility. The variability is due, in part, to the same uncertainties stated above. In addition, variability is introduced as a result of adjustments in the cost of the specific conventional-fueled engine used as a basis for comparison.
14. The economics of fueling a gas turbine plant with methanol are impacted most significantly by the cost of the fuel itself. The additional fuel cost of operating the UCD plant on methanol was approximately \$4,000 per day and reflected a \$5.00 per million Btu differential over natural gas.
15. The low emissions capability of methanol can be advantageous with respect to the cost of the fuel in cases where compliance with strict NO_x emission regulations is required. The higher cost of methanol may be offset by the costs associated with exhaust gas treatment equipment which may be required for a conventional-fueled turbine.

SCOPE OF WORK

The project comprised several specific elements and covered a wide range of technical disciplines. The work started with the conceptualization of the modifications necessary for fueling the UCD facility with methanol and continued through to the performance of the demonstration testing and analyses. The nature of the work covered within each element is summarized in the following paragraphs.

The facility was originally designed for operation only on distillate fuel oil. The existing fuel system and safety features of the plant were designed for the storage and handling and the code requirements of fuel oil. Since the characteristics of methanol are significantly different than fuel oil, design and fabrication of a methanol-compatible storage and handling system was necessary. Also, the existing plant safety features were not adequate for methanol and required augmentation. The major elements of the facility modification effort included: land preparation and foundations; storage tank installation; provision of a tank containment wall; design and fabrication of a fuel pumping, flow metering, and filtering module; design and fabrication of fuel receiving and transfer piping; and provision of water treatment and injection hardware. Ancillary systems included controls and lighting. The safety features of the plant were augmented to include: explosion proofing of electrical components; upgrading of ventilation systems; provision of methanol vapor and fire detection equipment; and various alarms.

Combustion hardware specifically optimized for the utilization of methanol in an Allison 501-KB gas turbine had not been developed at the onset of the program. In order to confirm that satisfactory combustion could be realized, Allison performed a series of combustor rig tests using methanol and water-in-methanol blends at their facility in Indianapolis.

The rig testing comprised several elements. The fuel injection nozzle design was modified to achieve the desired pressure/flow relationship. Acceptable ignition and flame stability characteristics were examined for various water injection rates. Emissions measurements were made to serve as a basis for comparison to data obtained on the UCD engine firing methanol.

Based upon the rig test data, Allison designed and fabricated all the components necessary to operate the UCD engine on methanol. These components

included the high-pressure fuel pump and filters, flow control valves, fuel dump and shut-off valves, fuel manifold, fuel nozzles, and all connecting piping and mounting hardware. Once the special methanol "kit" was completed, the components were installed on a 501-KB gas turbine at the Allison facility and operated to assure satisfactory operation prior to installation on the UCD engine.

The Allison methanol kit was installed on the UCD engine and the facility was run on methanol. Following a short period of operation, problems developed regarding the pumping of the methanol to the high pressures required for injection into the combustors. This unanticipated situation necessitated a major modification in the project scope and in the manner fuel was supplied to the engine. A task was undertaken by Allison to provide the necessary hardware redesign and fabrication to effect the reliable pumping of methanol to the engine. Following the implementation of the new pump system, the demonstration run on methanol was performed.

The demonstration testing performed by KVB was aimed at obtaining data to document the engine's emissions and operational performance during the operation on methanol. The testing was initiated by performing a series of parametric tests wherein NO_x and CO emissions were measured as a function of water injection rate and load. Once an optimum water injection rate was determined, the engine was operated for the remaining period at these conditions. During the extended run, measurements of other air pollutant species were made. The species of interest included particulate (total grain loading and size distribution) and aldehydes.

The original intent of the project was to obtain "baseline" emissions data with the engine operating on distillate fuel oil in order to enable a comparison between the two fuels. However, the engine was converted to natural gas operation, coincidentally within the same period as the demonstration, and no oil operation capability was retained. NO_x , CO, and aldehyde measurements were obtained, nevertheless, with the engine operating on natural gas as a basis for comparison.

The final element of the project comprised the analysis of all data obtained during the demonstration and the documentation of the results. The

analysis included both a technical evaluation of the demonstration results and an economic evaluation of the use of methanol as industrial gas turbine fuel.

DEMONSTRATION TEST RESULTS

The UCD gas turbine engine was operated on methanol for a total of 1,036 hours. The primary objective during this time was documenting the engine performance and emissions with the engine instrumentation and a continuous exhaust gas analyzer system. In addition, a limited number of tests were performed to determine aldehyde and particulate emissions. Periodic inspections were performed by boroscope observation of the engine hot sections to evaluate the condition of the engine.

Baseline Tests

Baseline (no water injection) emissions of NO_x , O_2 and CO_2 are shown in Figure S-1 for engine operation with methanol and natural gas. The natural gas tests were brief and were conducted to obtain comparative data for the more extensive methanol operation. Exhaust gas O_2 concentrations versus generator electrical output were nearly equivalent between the methanol and natural gas. As expected for a gas turbine, the oxygen content decreased with increasing load and higher fuel to air ratio. Similarly, CO_2 content increased with higher load, however a distinct difference is noted between the two fuels. Methanol has a 0.75 to 1.0 percentage point greater CO_2 content at all load settings. Considering that the magnitude of CO_2 in the flue gas was only 2 to 4 percent, this change is significant. The higher CO_2 was the direct result of the difference in combustion chemistry and carbon content of the fuels. Compared to natural gas, methanol has a higher carbon content, on an equivalent heat input basis.

Baseline NO_x emissions for methanol were significantly lower than for natural gas. Without water injection, operation on methanol resulted in emissions that were approximately one-third those for comparable operation on natural gas. Figure S-1 shows the NO_x emissions for methanol to range from 24 to 38 ppm (at 15 percent O_2) for a load range of 50 percent to full load. NO_x emissions were also calculated on a basis of micrograms per Joule output ($\mu\text{g}/\text{J}$) and are presented in Figure S-2. As shown, the program objective of

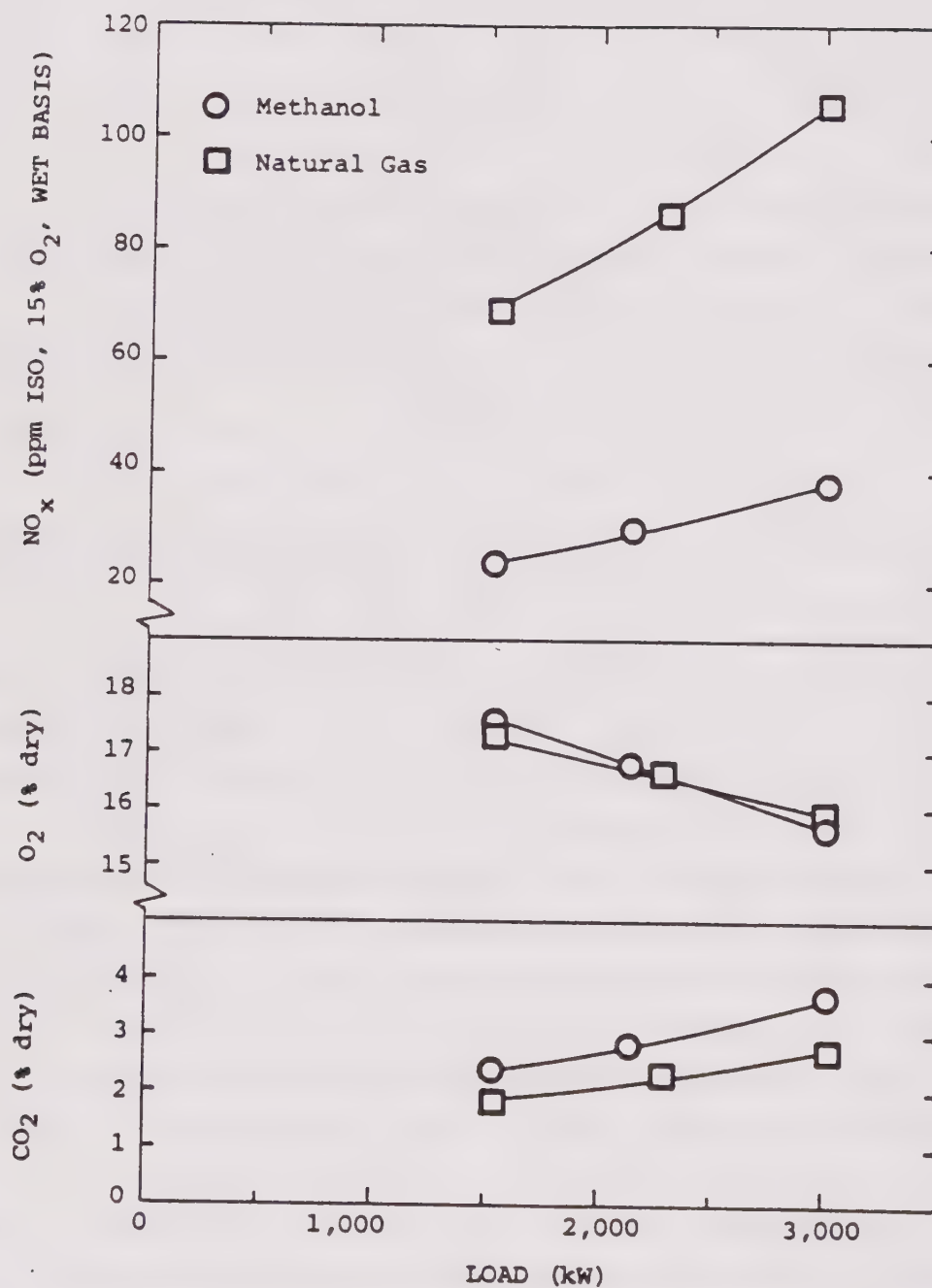


Figure S-1. Baseline emissions of NO_x, O₂, and CO₂ for methanol and natural gas operation, no water injection and variable load.

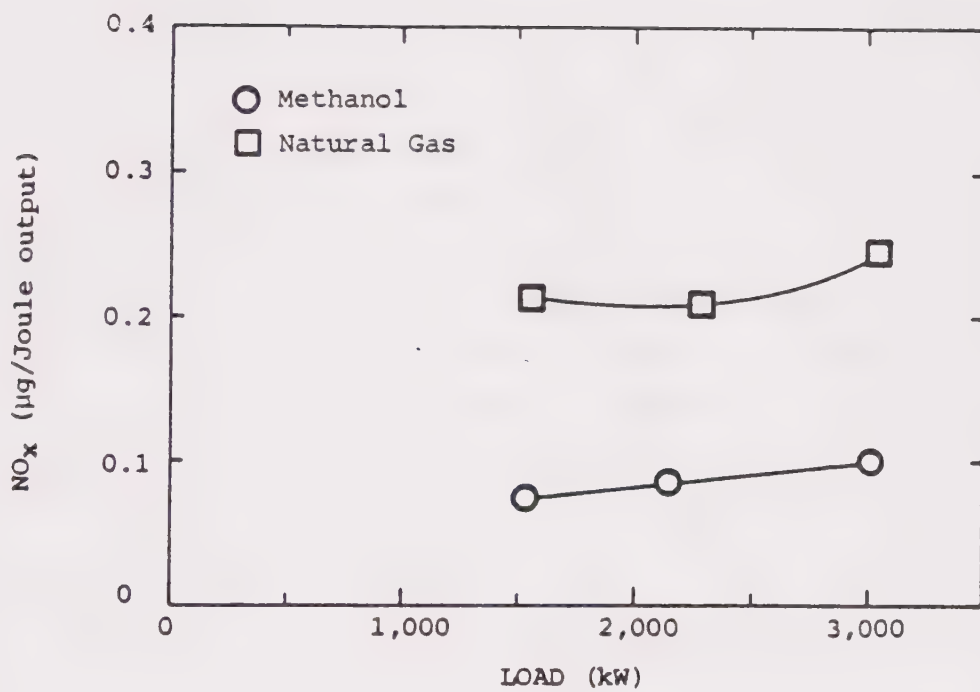


Figure S-2. Baseline NO_x emission for methanol and natural gas, operation, no water injection and variable load

0.1 $\mu\text{g}/\text{J}$ was met for methanol operation for all loads without the use of water injection. NO_x emissions on natural gas were between 0.2 and 0.25 $\mu\text{g}/\text{J}$ for the load range.

Combustibles, or incompletely burned fuel products, are generally represented by carbon monoxide (CO). For methanol combustion, aldehydes have also been found to be a significant combustible product. Figure S-3 shows CO and aldehydes emissions as a function of gas turbine load for baseline operation (no water injection) on methanol and natural gas. It is apparent that the CO and aldehydes (analyzed as ppm formaldehyde) emissions were related. Aldehyde emissions were significantly lower than CO emissions for both fuels. For operation on natural gas, the CO emissions remained at ~ 20 ppm throughout the load range, and aldehydes were relatively constant at ~ 0.2 ppm. At full load, the CO and aldehyde emissions for methanol were similar to natural gas. For methanol, however, the CO and aldehydes emissions increased as load was reduced. The fuel nozzles which had been modified for the higher flow rate capacity necessary for methanol operation may be partially responsible for the increase of combustibles emissions, due to less effective atomization at low flow rates. Additional research, development and optimization of the fuel nozzles and/or combustor liner may be necessary to improve fuel burnout at low loads, if these combustibles emissions are considered to be excessive. However, at the typical high load operation of a gas turbine engine, the combustibles emissions remained low for methanol.

Water Injection Tests

Water injection was performed only during operation on methanol as the natural gas installation at the UCD cogeneration facility was not so equipped. NO_x emission reductions with water injection are shown in Figure S-4. At 50 and 75 percent load, NO_x emissions were approximately 10 ppm with a water injection rate of 0.3 lb $\text{H}_2\text{O}/\text{lb}$ fuel, thus representing reductions of 58 and 66 percent, respectively, from baseline emissions. The maximum water flow rate for full load operation was limited to 0.24 lb $\text{H}_2\text{O}/\text{lb}$ fuel due to system flow capacity limits, however NO_x emissions were reduced to approximately 14 ppm, or a reduction of 63 percent from baseline. The 10 to

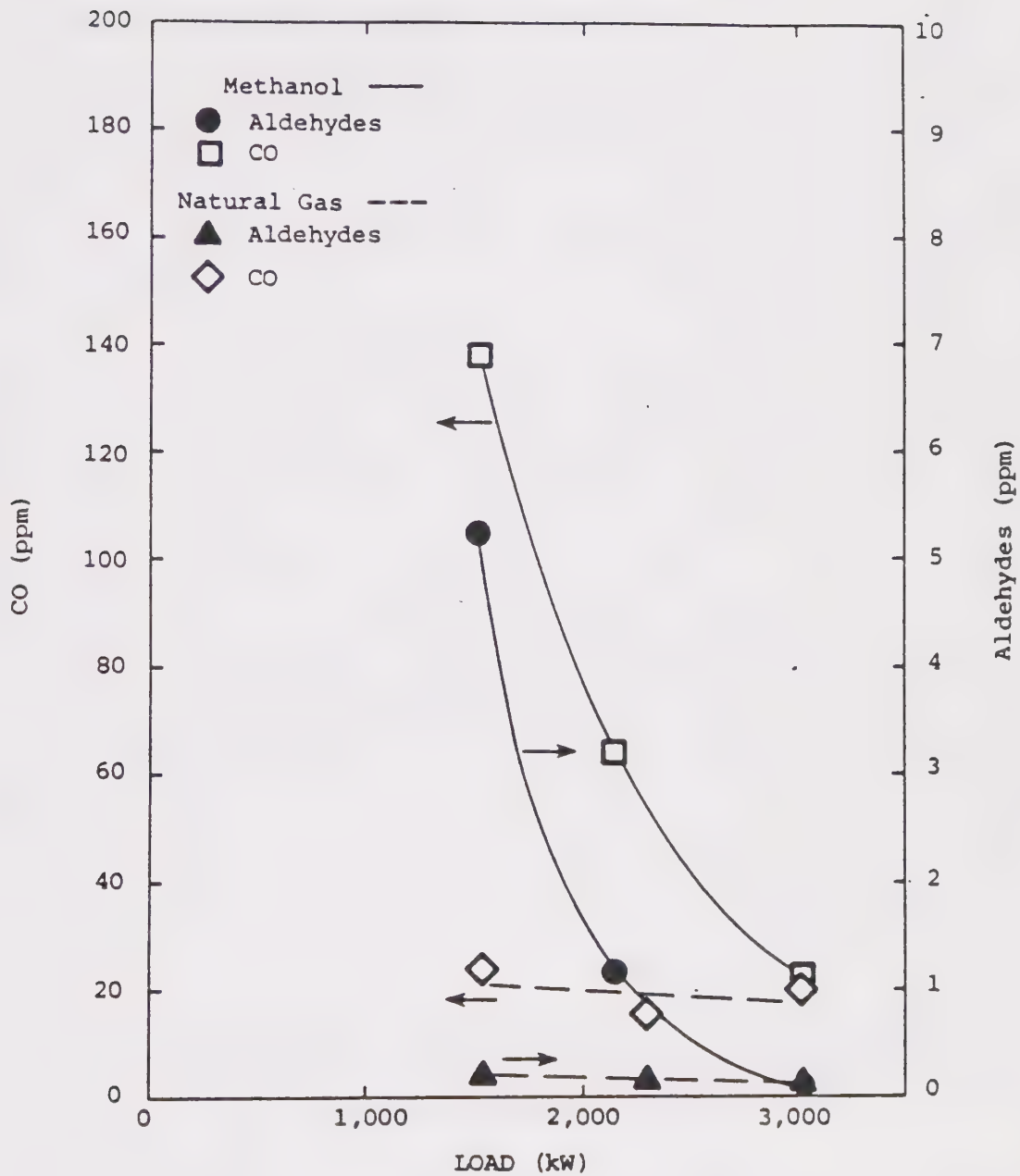


Figure S-3. Aldehydes and CO emissions for methanol and natural gas operation, no water injection and variable load.

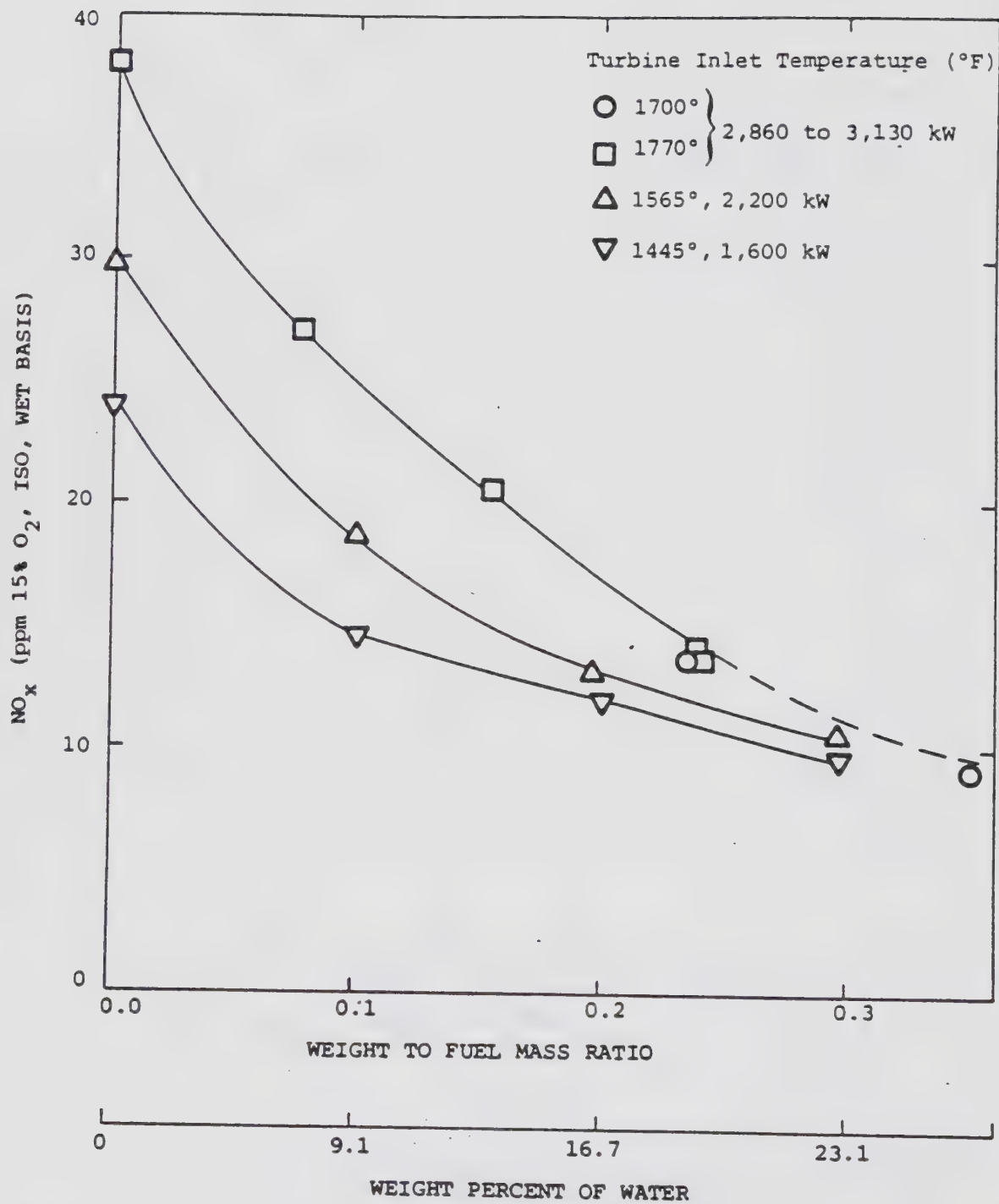


Figure S-4. Effect of water injection upon NO_x emissions (ppm)

14 ppm emission levels achieved with water injection on methanol represent 80 to 90 percent reductions compared to baseline natural gas NO_x emissions.

As with the baseline tests, combustibles (CO and aldehydes) emissions trends remained related (Figure S-5). Water injection influenced combustibles emissions during the low load tests, while emissions at full load were not affected and remained low (20 ppm CO and ~ 0.05 ppm aldehydes). At 50 percent load, CO and aldehydes increased 20 ppm and 1.5 ppm respectively, due to water injection. These changes were low in comparison to the effect of engine load upon combustibles.

Particulate emissions were measured at the full load and maximum water injection (0.24 lb H₂O/lb fuel) test point. EPA Method 5 emissions were below 0.01 lb/10⁶ Btu and comparable to emission factors which have been reported by the U.S. EPA for natural gas-fired gas turbines.

System Operation and Engines Inspections

The operability of the engine on methanol was noted to be satisfactory and equivalent to operation on natural gas. Few operational problems were noted during the test program. These included a flow induced vibration at low engine load and rust deposition in fuel filters. The vibration emanated from the engine-mounted fuel flow control valves and the piping returning excess methanol back to the centrifugal pump. Allison speculated that an optimized fuel system and engine control system would circumvent this problem.

Rust deposition on the fuel filters was an operational problem which resulted in excessive pressure losses and limited fuel flow rates. The rust was found to be originating from the methanol storage tank. Larger capacity, smaller particle size capture filters would be expected to sufficiently reduce the impact of the rust.

Boroscope inspections were performed before, during, and after the methanol demonstration by Allison representatives. These inspections revealed that the cross-over tubes between combustor liners (these allow ignition of one combustor to another) and a reverse flow baffle on the combustor liner were burning or eroding with time. The heat erosion of both areas of the combustor were described as minor and were considered within serviceable

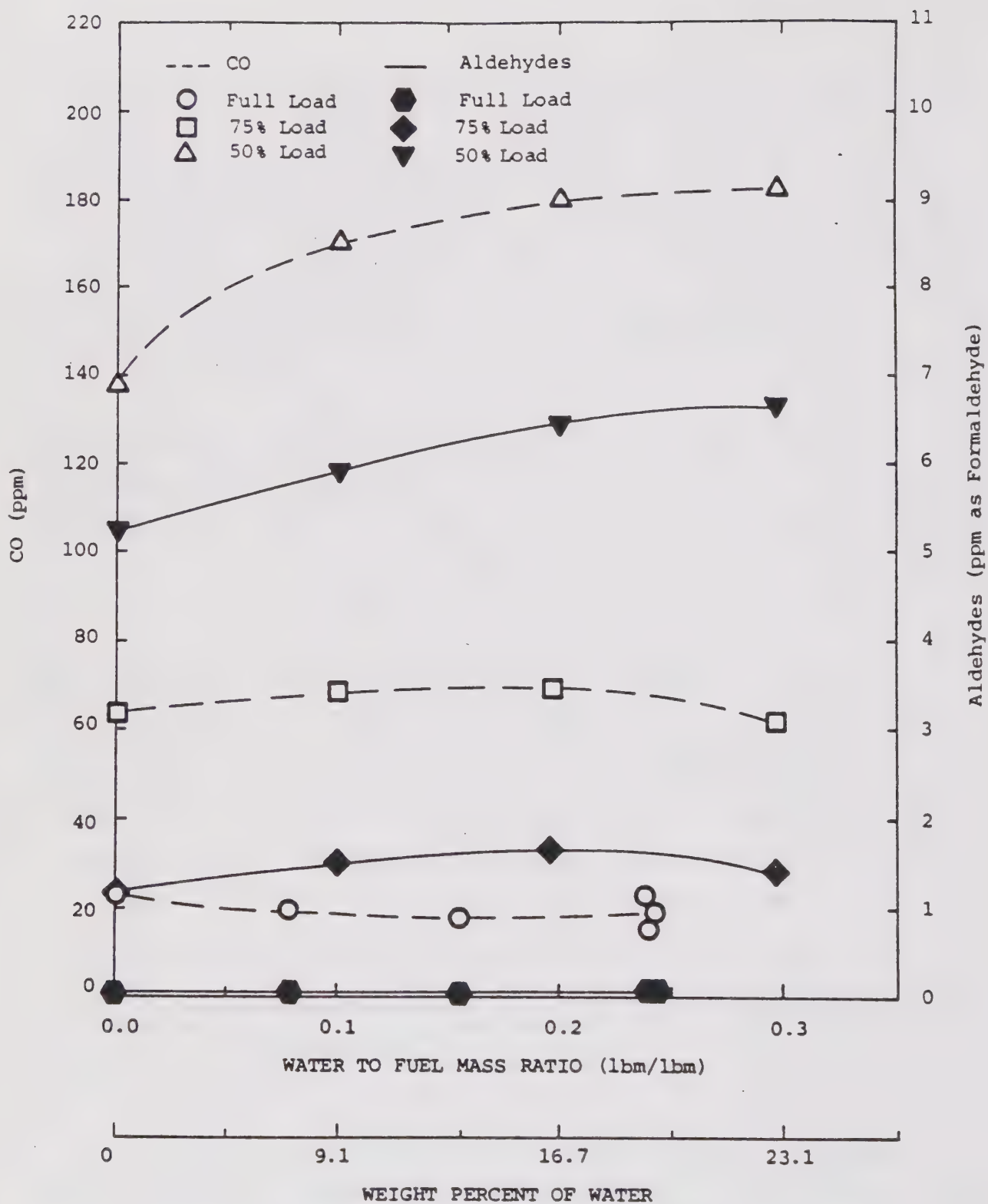


Figure S-5 Effect of water injection upon CO and aldehydes emissions.

limits for engine operation. The burning of the cross-over tubes was not detrimental to engine operation and is a condition typical for natural gas and liquid fueled engines of this design.

At the end of the methanol test program, the only significant changes were the continued burning of the aforementioned areas. All boroscope inspections showed the turbine blades and vanes to be in good condition, with no notable effects from the use of methanol. A reddish-brown color on the blades indicated that some of the rust was passing through the filters and a portion had been deposited on the blades. Although no detrimental effect was attributed to the rust coating, it was recommended that improved filtration be used in future applications to remove the rust.

During the last engine inspection following the completion of the test program, an internal engine oil leak (unrelated to methanol operation) was noted. The engine was removed and during the repair disassembly, over-heating damage to the second stage vanes and foreign object damage on the fourth stage blades of the turbine power section was found. By the nature of the damage, Allison determined that the turbine had been subjected to an over temperature condition during a startup, causing typical burning of some of the second stage vanes. Some of the metal thus burned away subsequently damaged the fourth stage blades (foreign object damage). The second stage vanes comprised both a hollow (older) and a solid, more durable (newer) design. Only the older, hollow style vane design was heat damaged. The overtemp condition was severe enough to damage the older less durable design, but not the newer vanes.

The damage was not observable with the equipment utilized for the boroscope inspections. Therefore it could not be determined when the damage had occurred; if it had occurred prior to the demonstration on natural gas, between methanol runs when the turbine was operated on natural gas, or during the demonstration testing. The damage was of a nature which, according to Allison, is not atypical on gas or oil operation. Thus, the damage was not necessarily the result of methanol use, although it could have been. Of significance, was the fact that although the time, fuel, or reason associated with the startup overtemp could not be determined, the latest design second

stage vanes were not damaged. Had the engine contained only the newer, solid vane design, no damage to the turbine would have occurred.

ECONOMICS

The capital costs to retrofit the UCD facility to methanol operation totalled \$670,000 and necessarily reflect the developmental nature of the program and the design/fabrication of a one-of-a-kind fuel system. Based on the cost information gathered during the program, the cost to retrofit a similarly sized (3,000 kW) commercial installation would be within a range of \$420,000 to \$769,000. This estimate is variable since the actual costs would be dependent upon the desired quantity of on-site methanol storage, whether the plant had been set up originally for gas or oil firing, and whether the existing design included a water injection system for NO_x control.

For a new installation of similar size, the capital cost difference between a methanol-fueled and a conventional-fueled plant is estimated to be within a range depending upon those aspects mentioned above and upon which conventional fuel is used as the basis of comparison. The difference could be as low as approximately \$18,000, or essentially zero within the estimation accuracy of the present analysis. At the upper end of the range, a new methanol-fueled plant could cost as much as \$497,000 more than a conventional fueled plant.

Based on the UCD experience, the operating and maintenance costs associated with fueling a gas turbine facility with methanol are similar to those of a conventional-fueled plant, in terms of operating labor, maintenance labor, and maintenance material costs. However, the overriding impact upon the operating costs of a methanol-fueled plant would be the cost of the fuel itself.

The methanol utilized for the demonstration at UCD was supplied at a delivered price of \$0.60/gallon which is equivalent to \$9.28/10⁶ Btu (HHV). Natural gas available to the facility during the demonstration ranged in delivered price from \$5.24 to \$5.40/10⁶ Btu. This difference translates to a daily total fuel cost differential of approximately \$4,000, based upon operating 24 hours per day at baseload (3,000 kW). A fuel cost differential

of this magnitude would likely be a detriment in the economics of fueling a gas turbine plant with methanol. However, provided that the demand for methanol increases and the appropriate manufacturing and distribution facilities are expanded, it is reasonable to speculate that the price of methanol may decline. Furthermore, the methanol used at UCD was a chemical grade product. The manufacture of lower purity methanol, suitable for gas turbine fuel, may also be expected to result in reduced fuel costs.

Another very important aspect concerning the cost of methanol as a gas turbine fuel relates to the low air pollutant emissions characteristics of the fuel. Certain air pollution control agencies require that NO_x emissions from gas turbines be controlled to levels of 25 ppm or less. In order to meet this level of NO_x emission on a natural gas- or distillate oil-fired turbine, water injection and a selective catalytic reduction (SCR) unit would be required. An SCR unit would not be necessary for a methanol-fueled gas turbine. The cost differential between methanol and natural gas or distillate fuel oil may be significantly offset by cost savings resulting from elimination of the SCR requirement.

METHANOL DUAL-FUEL COMBUSTION

by

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ABSTRACT

Dual-fuel combustion is a technique invented to lower emissions of NO_x from boilers. This paper presents experimental data using this technique in a utility boiler at a level of 35 megawatts. Tests with low sulfur (0.21% S) oil/natural gas, low sulfur oil/methanol, and natural gas/methanol were performed, and the NO_x emissions data were compared with NO_x emissions of 100% low sulfur oil, gas, and methanol.

Tests with a mixture of 70% natural gas/30% methanol revealed that the NO_x emissions level using the dual-fuel technique was lower than those obtained with either 100% gas or 100% methanol. The NO_x level was about 25% of the level obtained with natural gas fired in a conventional combustion mode or about 50% of the NO_x level obtained with natural gas fired in a "staged" combustion or "burners out-of-service" mode, a state-of-the-art combustion modification technique used to lower NO_x emissions.

DESCRIPTION OF EXPERIMENTS

1. BASELINE TESTS

Figure 1 is a sketch of the burner arrangement. In the data shown in Figure 2, the same fuel was combusted in all six burners.

Figure 2 presents NO_x emission data as a function of load for natural gas, low sulfur (0.21% S) oil, and methanol. The data points above 35 MW were obtained from Weir, et al.,⁴ in 1981 on the same boiler. The remainder of the experiments presented in this paper were performed at 35 MW. The 35 MW load was selected so that a wide range of fuel mixture ratios could be used. For example, it would be extremely difficult, if not impossible, to provide 80% of the fuel needed to generate 45 MW through only Burners 4, 5, and 6 (Figure 1).

2. "STAGED" COMBUSTION TESTS

Figure 3 shows the effect on NO_x emissions of "staging" with all gas fuel. By taking the No. 2 burner out-of-service (i.e., "staging") NO_x emissions are lowered in the case of gas by almost 50%.

Figure 4 shows the same type of "staged" combustion with low sulfur oil; however, the NO_x reduction in emissions is not as great as with gas.

In Figure 5, data is presented on three fuel combinations in the "staged" combustion mode - oil with No. 2 burner out-of-service, gas with No. 2 burner out-of-service, and a single point with a mixture of 70% gas and 30% methanol with the No. 2 burner out-of-service. In this latter experiment, methanol was fired in Burners 1 and 3 and gas in Burners 4, 5 and 6. Thus, while removing the No. 2 burner from service provides a reduction of about 50% in NO_x emissions with all gas fuel, the methanol over gas dual-fuel mode results in a further 50% reduction in emissions when the overall thermal input due to methanol was 30%. It should be noted, however, that there is a difference in burner equivalence ratio (i.e., stoichiometry) between the latter two cases.

3. DUAL-FUEL COMBUSTION TESTS

Figure 6 presents a comparison in which low sulfur oil is provided to Burners 4, 5 and 6 in both series of tests. In one case, gas is provided to Burners 1, 2 and 3 whereas in the second case methanol is provided to Burners 1, 2 and 3. The two curves cross over at about 70% oil thermal input.

The upper curve in Figure 7 presents the case in which methanol is fired in Burners 1, 2 and 3 and oil in the lower row of Burners 4, 5 and 6 (this curve uses the same data as the second case shown on the previous graph). In the lower curve, methanol is fired in Burners 1, 2 and 3 and natural gas in Burners 4, 5 and 6. It is important to note in both Figures 6 and 7 that by definition, a change in the percentage of fuel in both burner elevations results in a change of burner equivalence ratio for both burner elevations. This occurs because air flow to each burner remains constant.

DUAL FUEL METHANOL COMBUSTION

INTRODUCTION

One combustion modification technique used to reduce the amount of NO_x emitted in the boiler is known as a dual-fuel combustion process. This was discovered by Jones and Mansour^{1,2} during experiments in a 45 MW boiler at Southern California Edison's (SCE) Highgrove Generating Station with shale oil combustion. By burning a fuel oil which contained less nitrogen in the top row of burners over a shale oil containing a larger amount of nitrogen being combusted in the lower row of burners, NO_x emissions were lower than the NO_x emissions from burning a blend of the two oils. Later work performed by Radak,³ et al., with natural gas and low sulfur fuel oil in a larger boiler (175 MW) at SCE's Alamitos Generating Station confirmed the dual-fuel technique as a method in which lower amounts of NO_x could be obtained using this process compared to burning either of the pure fuels alone. During boiler tests (45 MW) in 1981 at the Highgrove Generating Station with pure methanol (Weir,⁴ et al.), a few experiments were performed with simultaneous combustion of methanol and gas in separate burners. E. A. Danko⁵ obtained a patent on the use of methanol and natural gas in the dual-fuel mode based on these experiments. Until the current work was performed, however, no test had been made of the dual-fuel concept with oil and methanol. These experiments, therefore, report the first use of methanol and oil in the dual-fuel mode and much more extensive experiments with methanol and natural gas in the dual-fuel mode.

Southern California Edison's Highgrove Generating Station Unit 4 is located in Grand Terrace, California, in San Bernardino County and was constructed in 1955. Highgrove Unit 4 is a drum type non-reheat boiler with a total of six burners in two rows as shown in Figure 1. The boiler is rated at 45 MW_e and was manufactured by Combustion Engineering. The burners used in the test program were Babcock and Wilcox "Racer" burners previously used in the 1981 pure methanol test. In the initial test of methanol combustion in a boiler in 1972 by New Orleans Public Service, and others, the use of burners not designed for methanol combustion resulted in many problems.

Modifications to the generating station necessary for methanol combustion were previously described.⁴ The same storage tank and the same rail car unloading facility used in the 1981 experiments were reused. The methanol pump was re-sized to permit accurate delivery of lower amounts of methanol. Consequently, maximum load capability on 100% methanol input was limited to 22 MW. Commercial methanol was supplied in four rail tank cars by Celanese Chemical Corporation. The higher heating value ranged from 9,390 to 9,500 Btu per lb. (21841 kJ/kg - 22097 kJ/kg).

STOICHIOMETRY IN DUAL-FUEL COMBUSTION TESTS

The symbol ϕ (or equivalence ratio) is frequently used to indicate the stoichiometry in combustion tests and is particularly convenient when different fuels are used since the absolute fuel/air ratio would be different at the stoichiometric point for each fuel. In this paper, ϕ is defined as the actual fuel/air ratio by weight divided by the stoichiometric or theoretical fuel/air ratio for that fuel. Thus, values of ϕ greater than 1.0 are fuel-rich while values of ϕ less than 1.0 are fuel-lean. (The assumption was made in calculating equivalence ratio that the air in the windbox was evenly distributed among the six burners.)

Figure 8 presents NO_x as a function of the equivalence ratio in the lower burners for various dual-fuel combinations. This illustrates the benefit of the fuel-rich combustion in the lower burner elevation and clearly indicates that methanol over gas allows lower NO_x emissions than methanol over oil or gas over oil. It was observed, at an equivalence ratio approaching 1.4 (corresponding to 20% thermal methanol input), that some difficulties were experienced with flame stability (this might not be true in larger boilers), but a satisfactory flame was obtained at an equivalence ratio of 1.3 (approximately 30% methanol input).

Figure 9 depicts in bar graph form, the benefits of dual-fuel combustion compared to gas and oil firing--both staged and unstaged.

DISCUSSION

The methanol dual-fuel process derives its NO_x reduction capability as the result of three factors. The first factor is that methanol contains no fuel bound nitrogen and, hence, no NO_x is formed from this source as in other conventional utility liquid fuels. The second factor is that the theoretical flame temperature is less than natural gas or fuel oil, thus less NO_x is formed by dissociation or the Zeldovich effect. The third factor is that fuel rich combustion of the primary fuel (natural gas or oil) results in less NO_x . It is well documented that fuel-rich combustion results in the formation of less thermal and fuel NO_x . The practical application of this process in a furnace (i.e., fuel rich combustion) is limited by the formation of partial oxidation products (CO) or soot (carbon or heavy hydrocarbons) resulting from incomplete combustion. Methanol allows fuel-lean combustion in the upper burner elevation providing vitiated air to complete the primary stage (gas or oil) fuel-rich combustion without producing substantial NO_x itself.

In the dual-fuel experiments, the thermal input due to methanol ranged from 20% to 55%. However, by comparison to 100% gas or 100% oil, it is possible to see the benefit in NO_x reduction obtained by substituting a little methanol for oil or gas in the top three burners. With oil, substituting 20% to 30% methanol, results in reducing the NO_x from 185 ppm to around 120 ppm (a 35% reduction). In an all gas-fired boiler, with all burners in service where the NO_x emissions level is around 215 ppm, substitution of methanol in the top three burners lowers the NO_x emissions level to slightly under 40 ppm. This is a little more than 80% reduction compared to gas being fired with all burners in service or about 50% of the level when the top center (No. 2 burner) is taken out-of-service (See Figure 5).

Figure 7 also indicates that substitution of 20%-40% methanol (as determined by fuel heating value input) in an all gas flame results in NO_x

emission levels which are lower than either 100% gas or 100% methanol without staging.

Since methanol, being normally produced from natural gas, is generally more expensive than natural gas, the desire to minimize the amount of methanol required to obtain significant NO_x reductions is evident. Thus, while the data indicates that 20% methanol would serve this purpose, flame stability and other operating problems dictate that 30% methanol should be the minimum amount of methanol substituted. This applies to the 45 MW boiler with only two rows of burners. It is quite possible that with larger boilers the minimum methanol input may be lower.

Figure 6 compares the effect of substituting either methanol or natural gas in the upper row of burners in an oil-fired boiler. There does not appear to be any significant difference whether natural gas or methanol is substituted for 15% to 35% of the oil being burned. Both cases result in around 40% NO_x reduction compared to 100% oil. For smaller thermal inputs of oil substitution, methanol is superior to gas substitution. The reason for the relatively flat NO_x curve with methanol over oil is not known.

The scenario for methanol use then is governed by fuel price and availability together with the need, if any, for NO_x reduction by the boiler operator. Referring again to Figure 6, one might say that since natural gas is cheaper than methanol, substitution of natural gas for 30% of the oil to achieve a 40% NO_x reduction would be the most economical path. However, one might question the likelihood of a situation in which 30%, but not 100%, of natural gas would be available for utility use at a single boiler. It would be possible, however, that methanol, a storable fuel, would be available when natural gas is not. In that case, a 30% methanol/70% oil use would result in a NO_x emission level of about 115 ppm, much lower than with 100 percent oil used in the normal manner. This level (115 ppm) is also lower than could be obtained by "staging" (taking No. 2 burner out-of-service) a 100% oil flame.

If natural gas, which may or may not be a cheaper fuel than low sulfur oil, were available, the substitution of 30% methanol results in greater NO_x reductions than could be obtained by "staging" the all gas flame.

One experiment was also performed with 100% methanol at a 20.5 MW load and No. 2 burner out-of-service. This resulted in a NO_x level of 32 ppm compared to NO_x levels of 43 to 57 ppm with no burners out of service at 20 to 21.5 MW. Thus staging of pure methanol allows a reduction in NO_x levels in addition to the reduction obtained by fuel switching (oil or gas to methanol).

FUTURE PLANS

As shown in Figure 1, the boiler used in these tests had only two rows of burners, three burners in each row. The tests were thus limited, since only one burner could be taken out of service and, as stated previously, there were limitations in how much conventional fuel could be "forced" through the lower three burners. To avoid these limitations, another set of dual-fuel tests with methanol are planned to be concluded during 1987 on a 215 MW boiler at SCE's Huntington Beach Generating Station. This boiler has four rows of burners, six burners per row so that some of the limitations of tests reported in this paper will be avoided. The tests are planned to be jointly sponsored by Celanese Corporation and Southern California Edison Company.

CONCLUSION

It is concluded that:

1. Substitution of methanol for 30% of natural gas (as measured by fuel heating value) results in NO_x emission levels considerably lower than either all gas, gas with a "burner out-of-service", or all methanol flames.
2. Substitution of methanol for 30% of the oil (as measured by fuel heating value) results in NO_x emissions levels lower than with a 100% oil flame, even if "staged". This option could be exercised if natural gas were not available but methanol, a storable fuel, was available and NO_x emissions levels were desired to be reduced.
3. Creating two discrete combustion stages within the boiler and selecting the combustion stoichiometry and fuel-type in each stage are key factors contributing to the NO_x reduction achieved by the process.
4. The NO_x reduction achieved by the dual-fuel process is the result of three factors: (1) Control of NO_x formation through the optimum use of fuel-rich combustion in the lower burner elevation, (2) Control of NO_x formation with the use of a nitrogen free fuel in the upper burner elevation, and (3) use of a fuel with a lower flame temperature.
5. There are no significant operating problems resulting from the use of dual-fuel methanol combustion when the local burner equivalence ratio does not exceed 1.4. At richer mixtures, burner stability problems may occur.

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LIST OF FIGURES
WITH CAPTIONS

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- Figure 7 - Dual-fuel combustion of methanol with oil and with gas at 35 MW. The top curve was previously presented as the lower curve in Figure 6.
- Figure 8 - NO_x emissions vs. burner equivalence ratio with dual-fuel combustion at 35 MW. All burners in service.
- Figure 9 - Comparison of NO_x emissions.

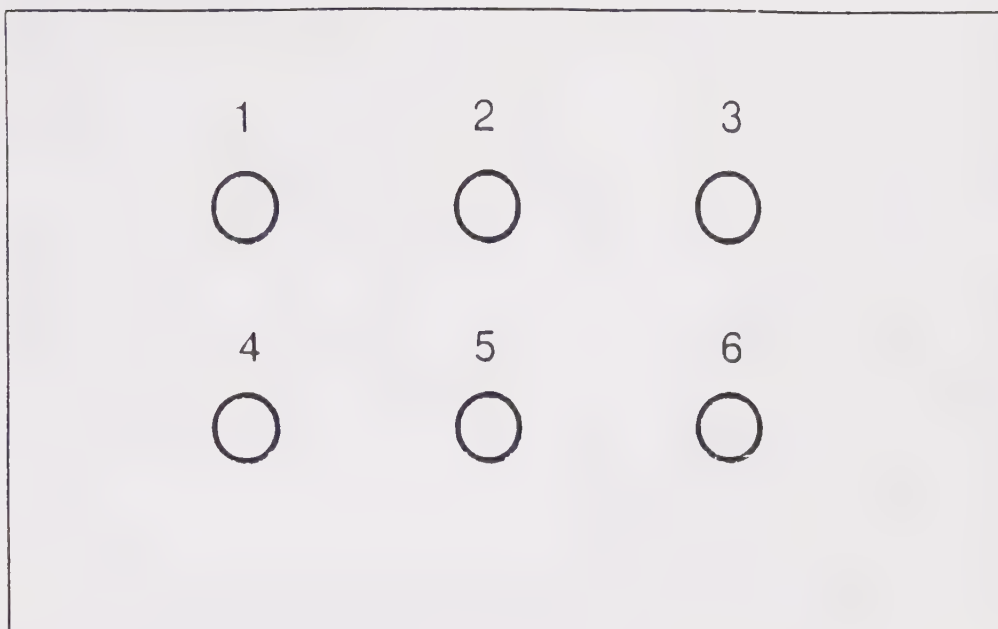


FIGURE 1: BURNER ARRANGEMENT - FRONT ELEVATION

NO_x , ppm Corrected to 3% O_2

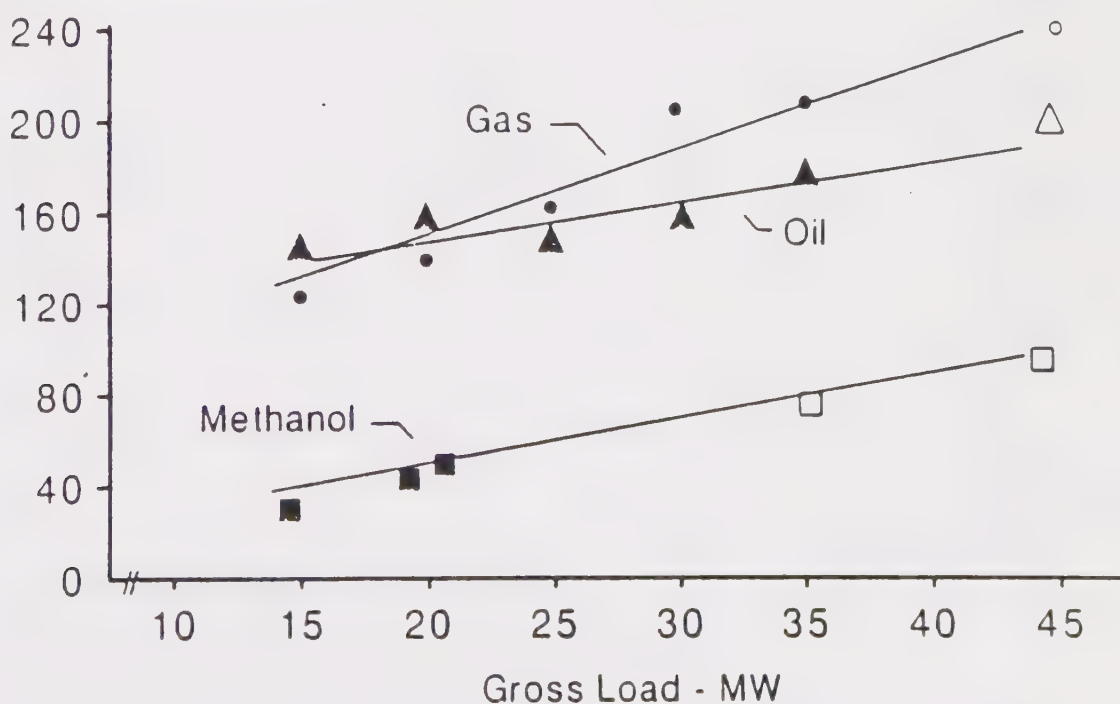


FIGURE 2: NO_x EMISSIONS AS A FUNCTION OF BOILER LOAD. ALL BURNERS ARE IN SERVICE. WHITE SYMBOLS REPRESENT DATA OBTAINED ON THE SAME BOILER IN 1981 (WEIR, ET AL)⁴

NO_x, ppm Corrected to 3% O₂

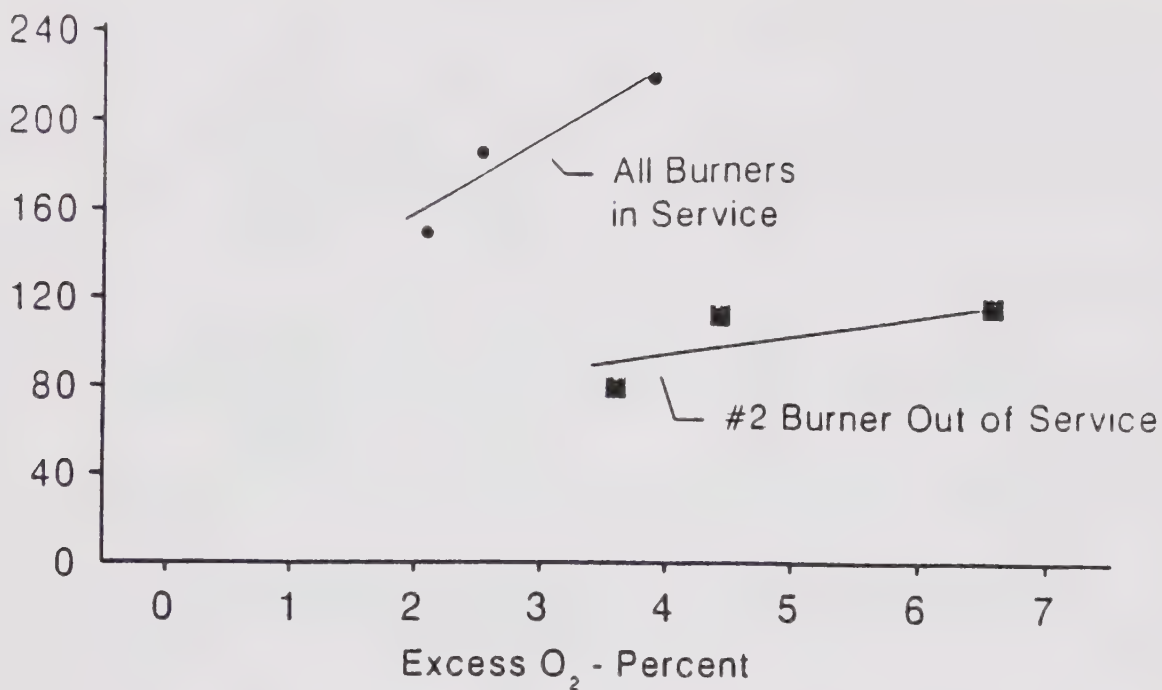


FIGURE 3: THE EFFECT OF "STAGING" WITH GAS FUEL AT 35 MW.

NO_x, ppm Corrected to 3% O₂

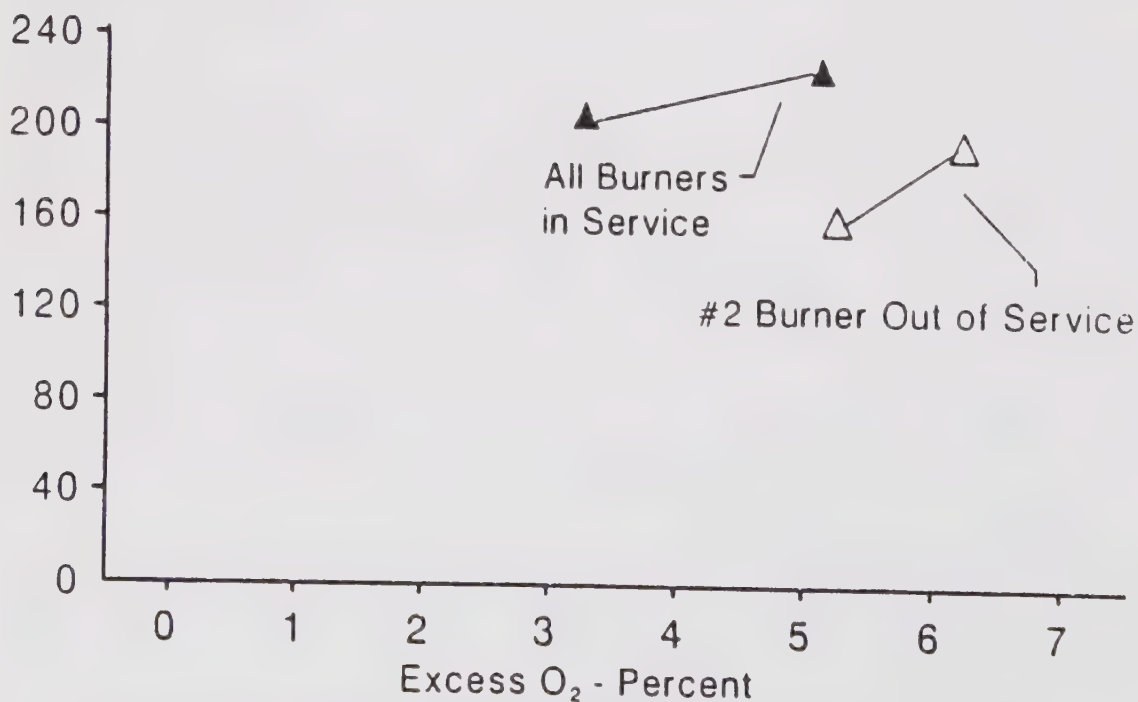


FIGURE 4: THE EFFECT OF "STAGING" WITH OIL FUEL AT 35 MW.

NO_x, ppm Corrected to 3% O₂

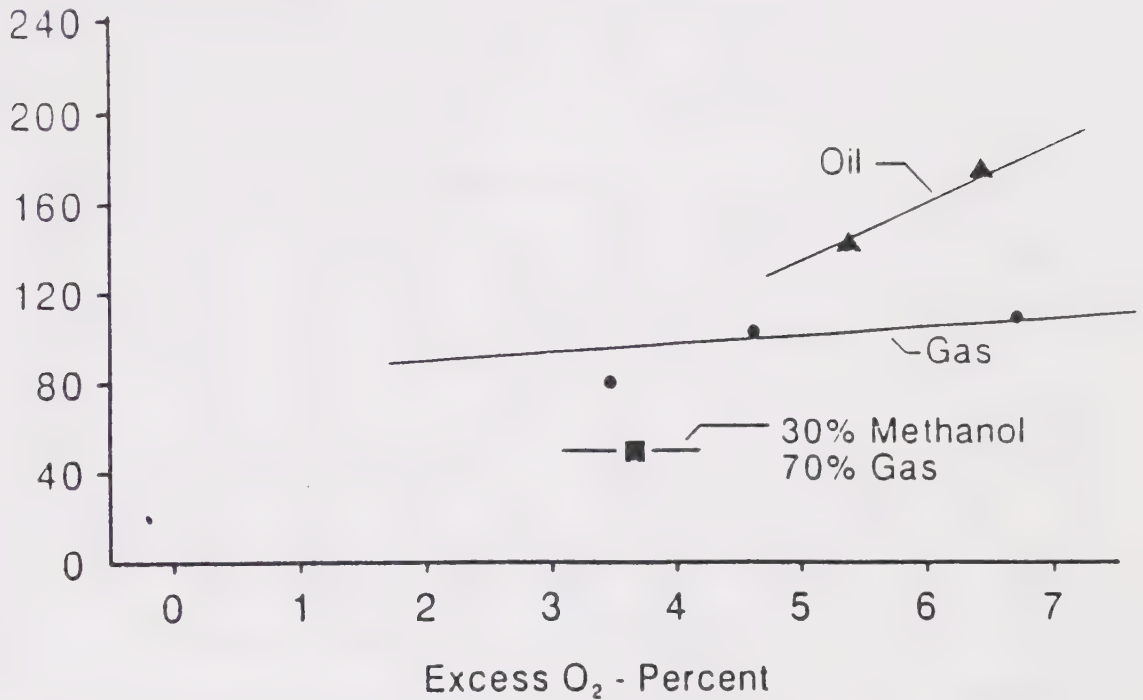


FIGURE 5: COMPARISON OF NO_x EMISSIONS WITH VARIOUS FUELS AND NO.2 BURNER OUT-OF-SERVICE AT 35 MW.

NO_x, ppm Corrected to 3% O₂

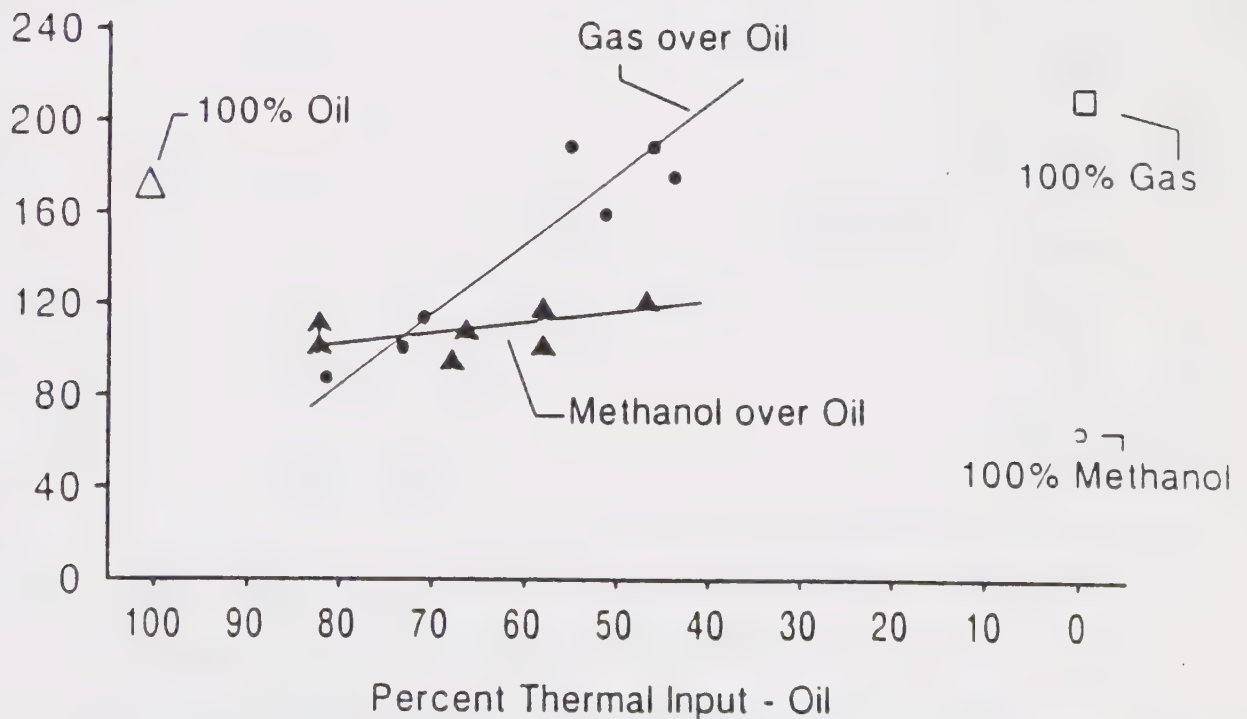


FIGURE 6: COMPARISON OF METHANOL AND GAS IN DUAL-FUEL COMBUSTION OIL AT 35 MW.

NO_x , ppm Corrected to 3% O_2

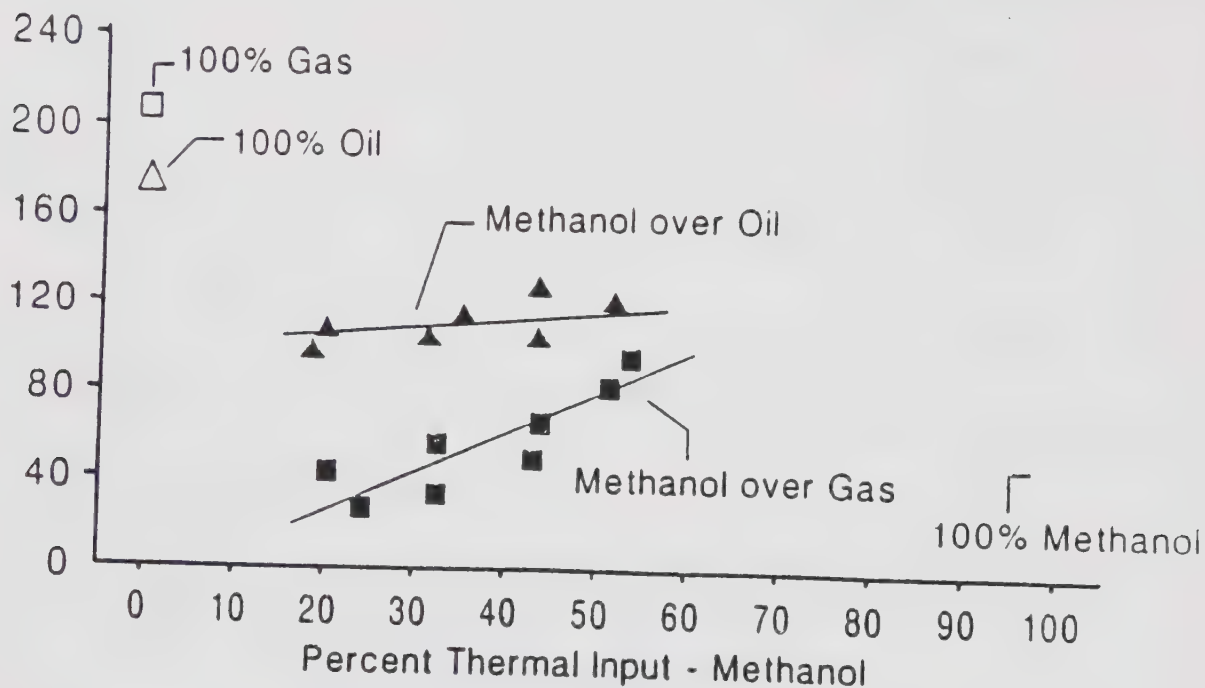


FIGURE 7: DUAL-FUEL COMBUSTION OF METHANOL WITH OIL AND WITH GAS AT 35 MW. DATA IN THE TOP CURVE WAS PREVIOUSLY PRESENTED IN THE LOWER CURVE IN FIGURE 6.

NO_x , ppm Corrected to 3% O_2

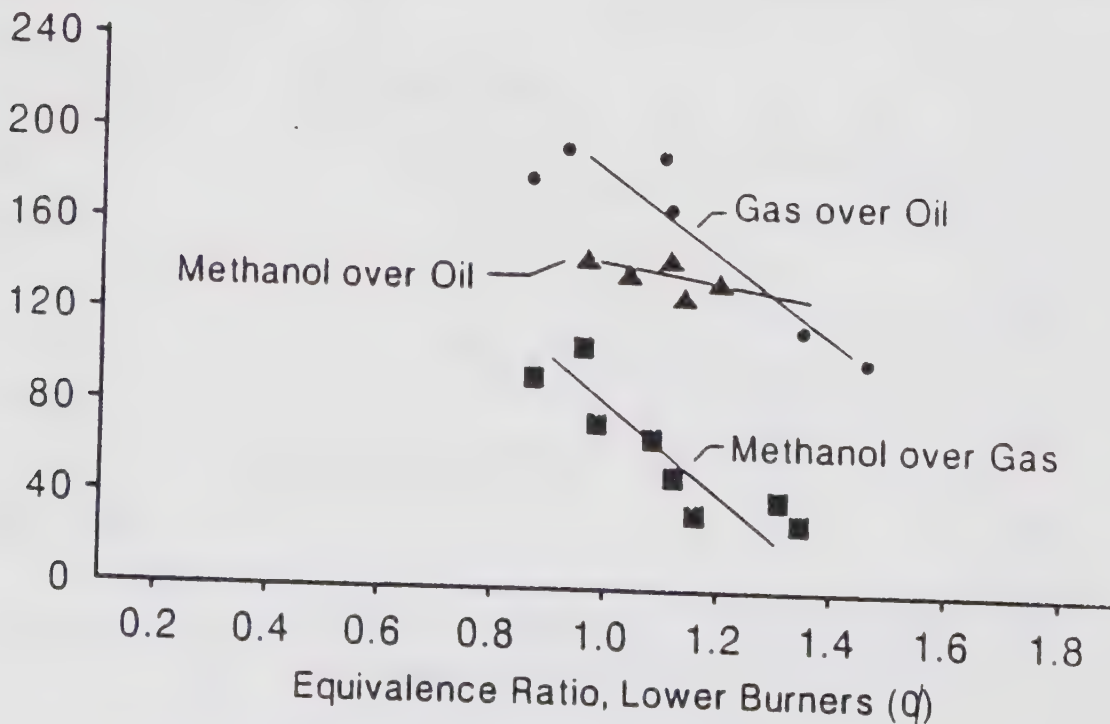


FIGURE 8: NO_x EMISSIONS VS BURNER EQUIVALENCE RATIO WITH DUAL-FUEL COMBUSTION AT 35 MW. ALL BURNERS IN SERVICE.

NOx PPM Corrected to 3% O₂

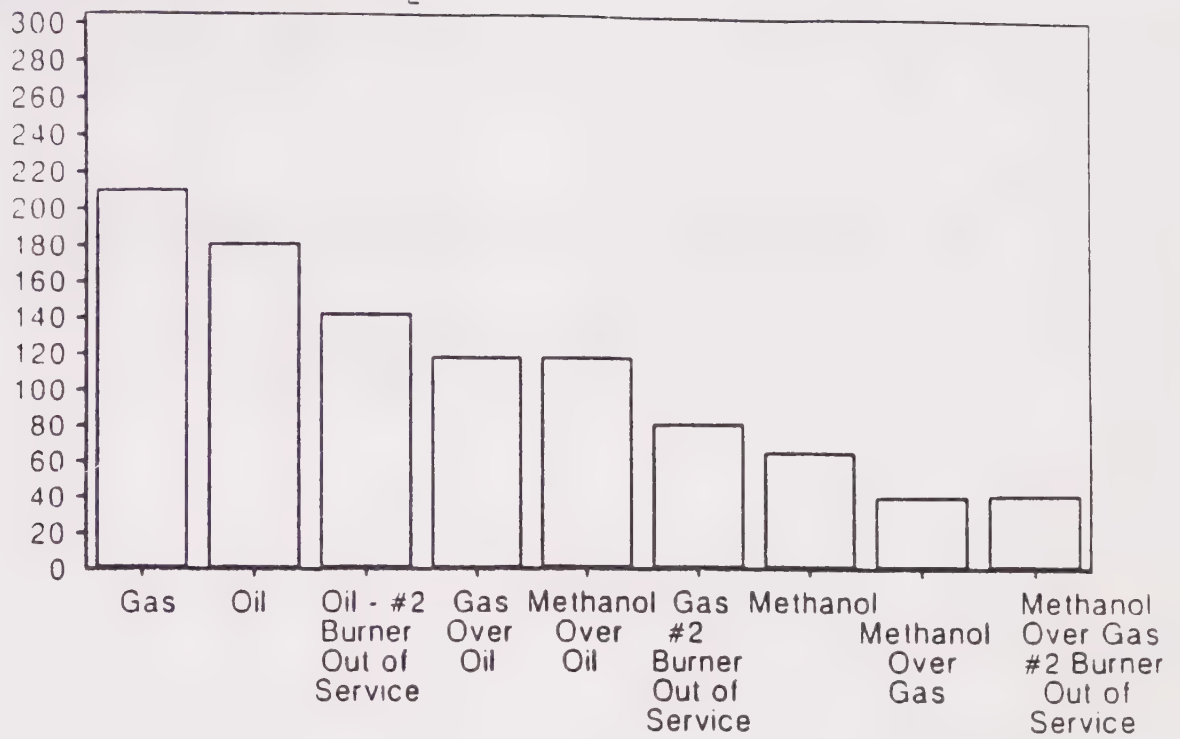


FIGURE 9: COMPARISON OF NOx EMISSIONS



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New Cycles for Methanol-Fuelled Gas Turbines

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ABSTRACT

Assuming that methanol is employed as fuel, the heat released from the gas turbine discharges can be used to cause an endothermic catalytic reaction:

$\text{CH}_3\text{OH} + \text{H}_2\text{O} + \text{heat} \longrightarrow 0,5 \text{ CO} + 0,5 \text{ CO}_2 + 2,5 \text{ H}_2 + 0,5 \text{ H}_2\text{O}$; this produces a gaseous fuel, the lower heating value of which exceeds that of methanol by 18%. Combining both steam reforming of methanol and steam injection in the combustor by using the maximum heat available in the exhaust gases, very interesting cycle characteristics can be achieved (more than 50% efficiency (LHV basis), same capital cost per kW as simple cycle gas turbine (9000E engine), low emissions of NO_x and SO_2). Reheating the gas during the expansion will improve the efficiency by 2 - 3 points allowing an increase in power output without increasing the capital cost per kW. At the end of the century, these types of cycles could be applied to all the new, non-nuclear power plants in the French energy system. The annual cumulative duration of such generators will not be greater than 2000 hours.

INTRODUCTION

In view of the probable future electricity consumption in FRANCE, the sharp increase in nuclear power plant unit capacity as well as the strict regulation concerning pollutant emissions (SO_2 , NO_x ...), the new thermal power plants that will be commissioned by the end of the century will have to meet the following requirements:

- low investment cost (annual hours of operation \approx 2000).
- great operating flexibility (numerous start-ups, and shutdowns, quick load variations).
- non-polluting.

Between fuel-intensive, simple-cycle gas turbines and capital-intensive, low-flexibility steam plants, there is room for new thermodynamic cycles using methanol, a clean, perfectly desulfurized fuel. By recovering heat from a gas turbine exhaust simultaneously to steam-reform methanol and produce steam for injection into the gas turbine, clean, high-efficiency cycles can be achieved at an investment cost close to that of a simple-cycle gas turbine.

1. POSSIBILITIES OFFERED BY METHANOL

With methanol - as opposed to combined cycles in which the gas turbine exhaust heat is used to heat up air or water - this energy can be directly supplied to the fuel.

Two methods can be used: evaporation and steam-reforming.

If evaporation takes place in a heat exchanger mounted at the turbine exhaust, the gas turbine efficiency can be increased by about 5% (the heat of vaporization for methanol is about 5% of its heating value). The fuel can be further converted by using a highly endothermic chemical reaction to recover the heat in the gas turbine exhaust, for instance through catalytic steam-reforming of methanol. Figure 1 shows the operating principle of such a cycle using a compressor of the type employed in the 9000E ALSTHOM gas turbine.

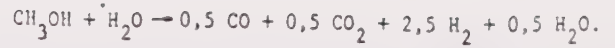
The chemical reaction whereby methanol is catalytically steam-reformed is the following :



The final composition of the gas mixture depends on the reaction temperature and the molecular ratio:

$$n = \frac{H_2O}{CH_3OH}$$

The amount of water supplied must be sufficient to prevent the formation of carbon black, while not exceeding a certain level if the catalyst efficiency is to be maintained. If the reaction ends at about 500°C, 20 bars and if a molecular ratio of $n = 1$ is used (compromise) the overall reaction is:



The gas mixture produced has a heating value 18% higher than that of methanol so that the cycle efficiency increases by about 18%. In this reaction, non-combustible gases (CO_2 and H_2O) are also introduced into the combustor. They will have the same effects as a steam injection, i.e. an increase in the gas turbine power. Indeed, these gases enter the combustor without passing through the compressor since methanol and water are compressed when liquid (less energy required). From a technological point of view, the steam-reforming reaction takes place in conventional heat exchangers at the turbine exhaust. The Cu-Zn-Cr catalyst is placed inside the finned tubes around which the turbine flue gases circulate.

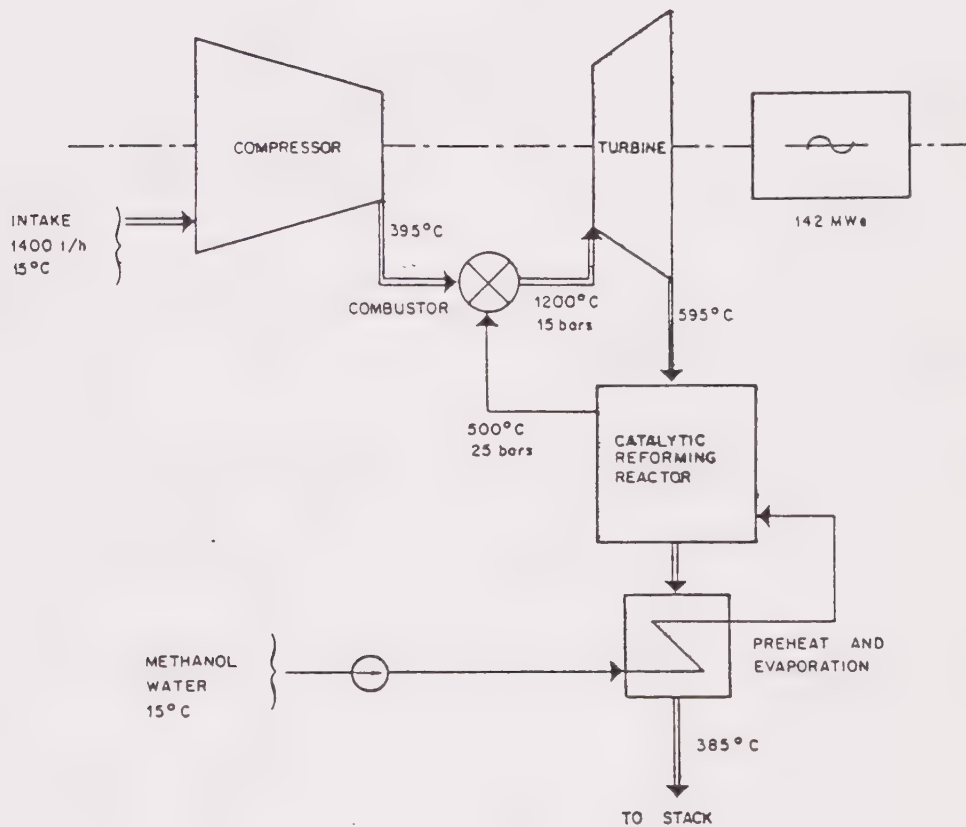


FIGURE 1: Cycle using methanol steam-reforming

The steam-reforming reactor is quite widely used to produce H_2 in small quantities at about $300^\circ C$. However, switching to larger capacities and different operating conditions (30 bars, $500^\circ C$) requires feasibility tests, which we are now performing in collaboration with a firm specialized in methanol

chemistry. This contract includes aging tests on the catalyst (risk of sintering at high temperatures), tests to optimize the H_2O/CH_3OH molecular ratio, and tests to determine the temperature gradients in the tubes and the reaction rates achievable in the reactor.

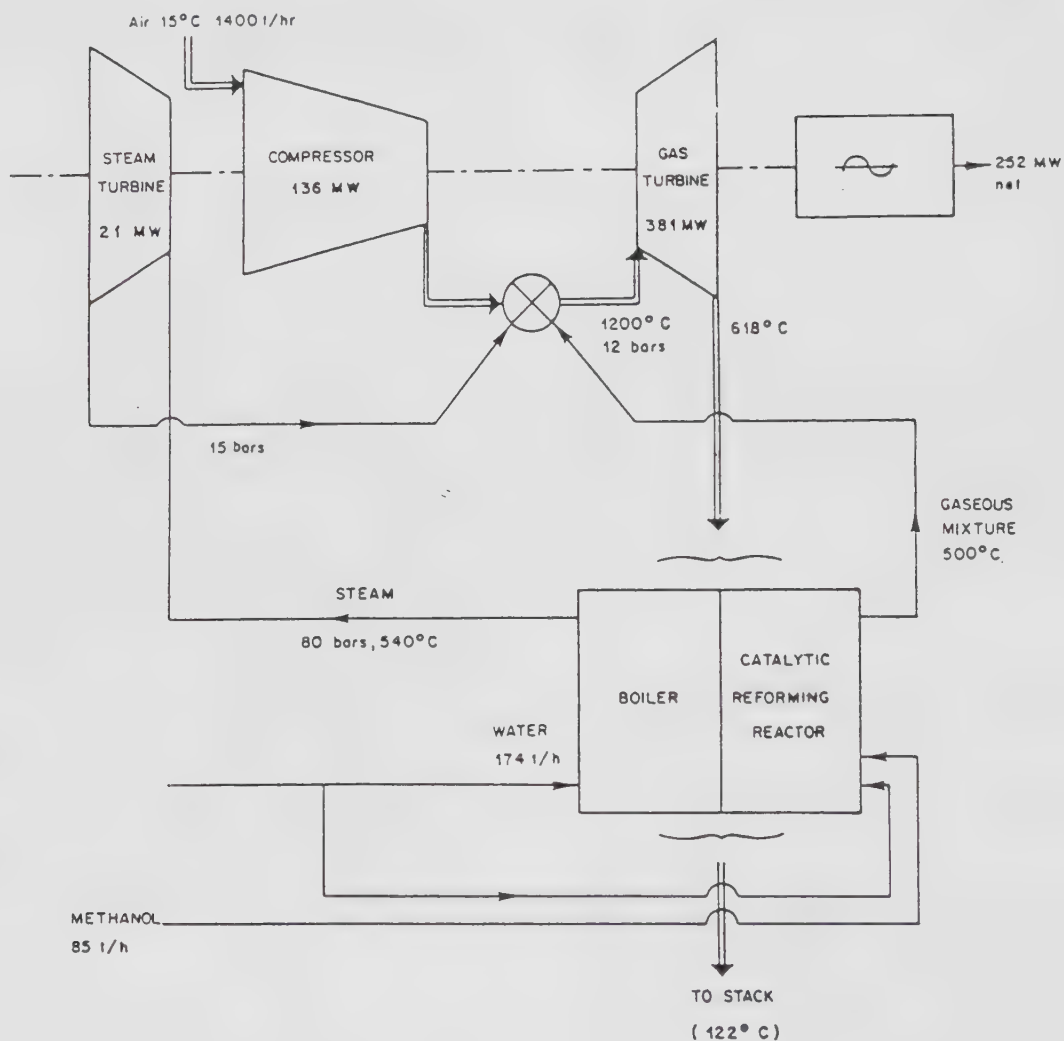


FIGURE 2: Steam-Reforming and steam injection cycle

2. STEAM INJECTION INTO THE GAS TURBINE

This step is used mainly to reduce NO_x emissions to the atmosphere, but the amounts of injected steam are small. If all the energy in the exhaust gases were to be recovered by producing steam for injection into the combustor, the steam flow would reach 29% (in terms of weight) of the air flow rate in the compressor. With such steam rates, it would certainly be more efficient to design a conventional combined cycle. Moreover, as soon as the steam-to-air weight ratio reaches 15%, a plume forms at the stack. Tests recently carried out by General Electric Company (reference [2]) have shown that, with a 7% steam-air ratio, the output power could be increased by 40%, the LHV efficiency by 14% and the CO and NO_x emissions could remain below 25 ppm (compared with a simple cycle with no steam injection).

3. METHANOL STEAM-REFORMING AND STEAM INJECTION CYCLE

The combination of methanol steam-reforming and steam injection makes it possible to recover all the heat available by injecting steam into the turbine with a steam-to-air ratio limited to approximately 9%. With the same air compressor as the one in the biggest heavy-duty turbine marketed to-day by ALSTHOM (9000E) and with the turbine inlet characteristics (1200°C, 12 bars), the cycle shown in Figure 2 is obtained.

Compared with a simple liquid-methanol cycle and with the simple steam-reforming cycle, the following figures are obtained :

	simple liquid methanol cycle	methanol steam- reforming cycle	steam reforming+ steam injection cycle
net power output (MWe)	134	142	252
LHV efficiency (%)	34	41	53,5
demineralized water consump- tion (l/kWh)	-	0,25	0,7
construction cost par kWe (simple cycle: 100)	100	108	85

Table 1: Comparison of methanol cycles using gas turbines

Thus, when the cycle with methanol steam-reforming and steam injection is used, instead of the simple cycle (see Table 2) :

- the net power output increases by 88%
- the turbine power output increases by 32% only
- the thermal power released in the combustion process increases by 15% only
- the compressor power remains the same.

However, the last three parameters are essential for investment cost evaluation. The variation in the main power components is smaller than that of the net power output so that the construction cost is lower even though the waste heat boiler is not taken into account. Moreover in this technical and economic study, we did not make allowance for the efficiency and power gains achieved when water vapor is used instead of air in the various compressor and turbine glands...

	gas turbine simple cycle	methanol steam-reforming + steam injection cycle	variation
compressor mechanical power	136 MWe	136 MWe	=
turbine mechanical power	289 MWe	381 MWe	+ 32 %
combustion thermal power	409 MWth	471 MWth	+ 15 %
net power output	134 MWe	252 MWe	+ 88 %

Table 2: Variation in the main power components

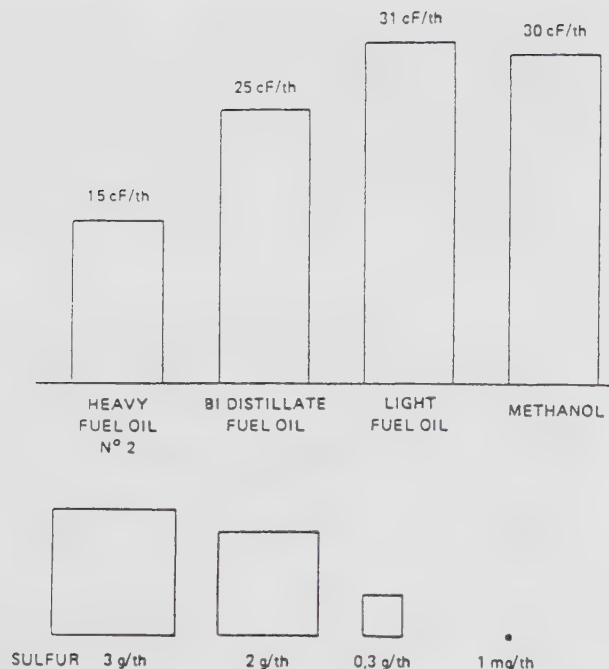
The other advantages of this type of cycle are :

- the absence of condensor enabling a plant using this system to be easily built near load centers or critical points of the transmission network.
- low SO₂ emissions thanks to the low sulfur content of the fuel (methanol: 1 mg/th against 300 mg/th for light fuel oil) (Figure 3).
- low NO_x emissions owing to the injection of steam into the combustor (contents below 50 ppm can be obtained).

On the other hand, demineralized water is required for this cycle. As a matter of fact, water purity is essential to avoid erosion-corrosion of the turbine blades (trace metal (Na, K...) concentrations must stay below 0.1 ppm).

In addition to the modification of the methanol steam-reforming process, the use of this cycle requires that the combustor will be adapted to a gas with a relatively low heating value (LHV of the order of 2 th/m³ compared with 9-10 for natural gas) and the injection of a significant amount of steam.

The overall operation of this cycle (start-up, shutdown, transients) should also be studied as well as the lineshaft stability (steam turbine, gas turbine, compressor, alternator).



NB : Prices given above are estimated for the end of the century

FIGURE 3: Fuels for peak load power plants ~

4. METHANOL STEAM REFORMING, STEAM INJECTION AND REHEAT GAS TURBINE CYCLE

As a result of the development of the preceding cycle it appeared that the size of the turbines themselves should be increased. Therefore a new rotating machine should be designed, which might take a long time and be costly. Moreover, this STIG (Steam Injected Gas Turbine) can only be mass-produced when it is also capable of burning bidistillate or light fuel oil or methane in good conditions: with the preceding cycle, to be able to switch from methanol to fuel oil or methane, the steam-reforming reactor must be converted into a steam boiler and combustors accomodating an injection of steam amounting to 30% of the air weight must be developed.

Gas reheat during the expansion could be used as a means of avoiding unit power increases and postponing innovations on static equipment by laying out rotating machinery in a different way (see Figure 4).

However, in this case 2 lineshafts exist:

* The first lineshaft (shaft # 1) is made of:

- an air compressor with a flow rate lower than that in the biggest General Electric compressor (60 Hz).
- a back-pressure steam turbine that can start the entire turbine generator.
- a gas turbine (# 1) corresponding to the HP stage (expansion interrupted at 6 bars) of the 60 Hz General Electric gas turbine. The latter has multiple General Electric combustors (combustor # 1 in Figure 4) that should be adapted to the injection of water vapor amounting to 15% of the air weight and to the combustion of LHV gas (2.3 th/m³).

This lineshaft # 1 is of the variable-speed type so that the thermodynamic efficiency at prolonged partial loads can be improved and the compressor and part of the 60 Hz General Electric turbine can be used.

* The second lineshaft is made of:

- a single combustor fed with the exhaust gas from gas turbine # 1 and with air-conditioning water vapor, which is warmed up before it is injected into the multiple combustors of gas turbine # 1. The development of this stationary equipment operating at a pressure of a few bars and at a high temperature should not raise particular difficulty.
- gas turbine # 2 coupled to a 50 Hz alternator. As this lineshaft is very short, a twin cylinder gas turbine (1200°C, 3000 rpm) with cylinders of the maximum achievable size considering present state of the art could be used and a dual inlet nozzle could be developed. Existing rotors and stators should also be adapted to the divided expansion. The difference of temperature existing in the exhaust gas of gas turbine # 2 (figure 4) could eventually allow the use of an air heating exchanger or a methane-reforming reactor if it is used as fuel instead of methanol.

Thus, this new cycle including methanol steam reforming, steam injection and gas reheat, which at first looks very sophisticated, is in fact only a new arrangement of existing rotating machines using stationary components which are still to be developed. A first technical and economic study yielded LHV efficiency above 55% and construction costs similar to those of the steam-reforming and steam injection cycle presented in paragraph 3°.

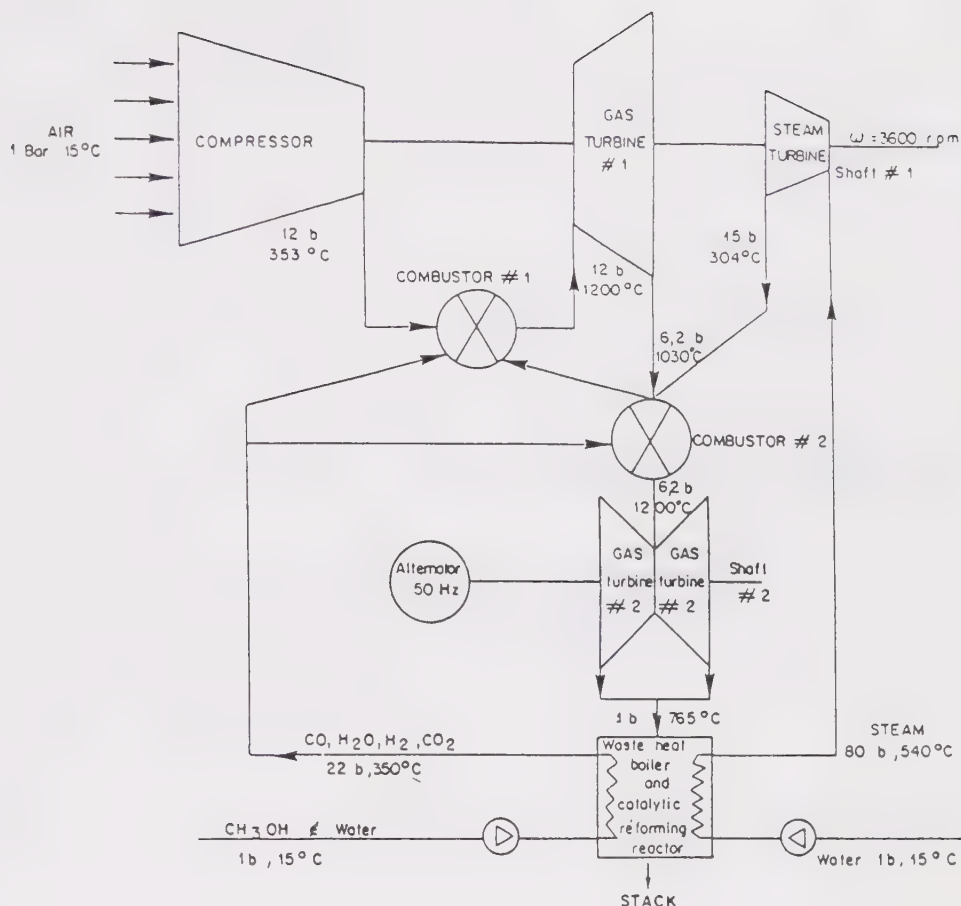


FIGURE 4: Methanol Steam-Reforming, steam injection and reheat gas turbine cycle.

5. METHANOL SUPERIORITY AS A FUEL

Besides the fact that its combustion is clean and that the power output can be increased by 8% when it is used instead of distillate fuel oil, methanol has a number of economic advantages. It is still difficult today to produce comparative methanol and fuel oil costs because of the uncertainties and tensions existing on the oil market. Nevertheless, a maximum cost of about 25 to 30 cF/th could be

postulated for methanol derived from coal in large quantities. Moreover, the cost of methanol covers mainly fixed carrying charges and operating costs so that one can reasonably assume that the price of methanol will be more insensitive to market fluctuations. Finally there are several ways of producing methanol, all at rather similar costs,

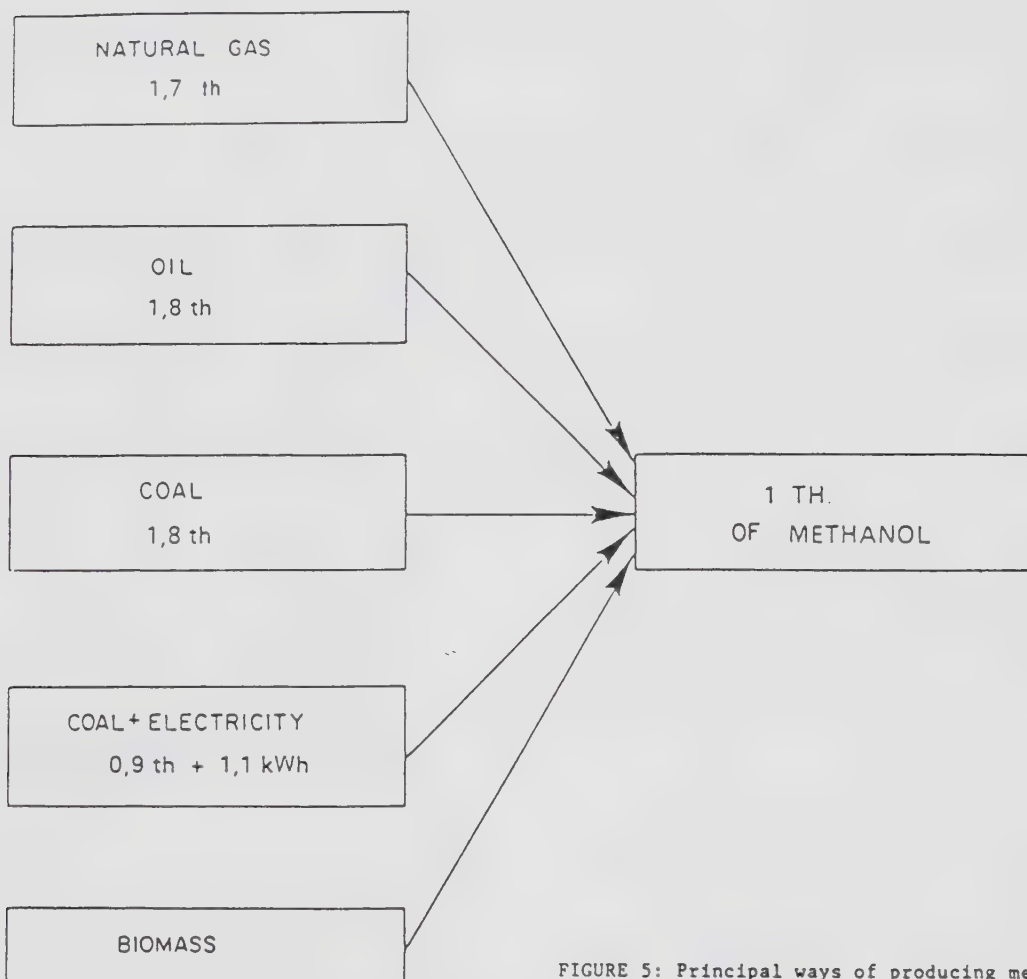


FIGURE 5: Principal ways of producing methanol

(natural gas, oil, coal, biomass, gas recovered from offshore oil fields and iron and steel industry...) (figure 5). Among the methods for producing methanol from coal, one uses coal only (figure 6). In this process, the oxygen required to gasify coal is generated in an oxygen plant and the gas is enriched with hydrogen by shift conversion $\text{CO} + \text{H}_2\text{O} \rightarrow \text{CO}_2 + \text{H}_2$.

In the combined coal/electricity process (see figure 7), the two oxygen production and shift conversion operations are replaced by a single operation - water electrolysis supplying both the oxygen needed in the gasifier and hydrogen for methanol synthesis, thanks to the use of cheap nuclear power produced during off-peak hours. Thus whenever the coal costs significantly fluctuate and methanol becomes expensive, the high coal costs are offset by using electrical power instead so that one might expect that methanol prices should not exhibit such large fluctuations as fuel oil prices.

FIGURE 6: Methanol production by the coal process
(oxygen production + shift conversion)

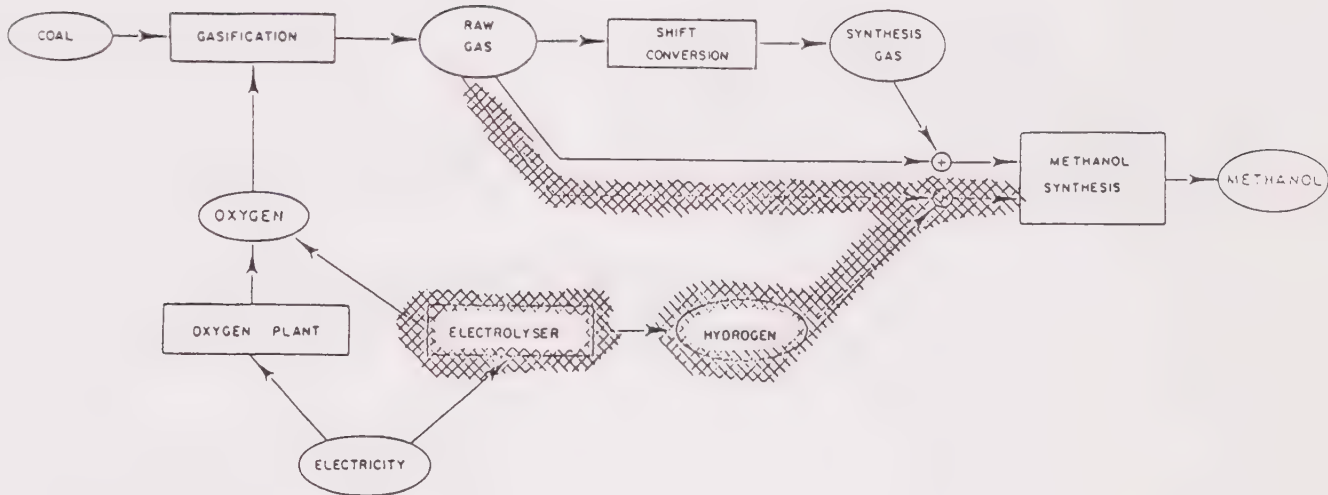
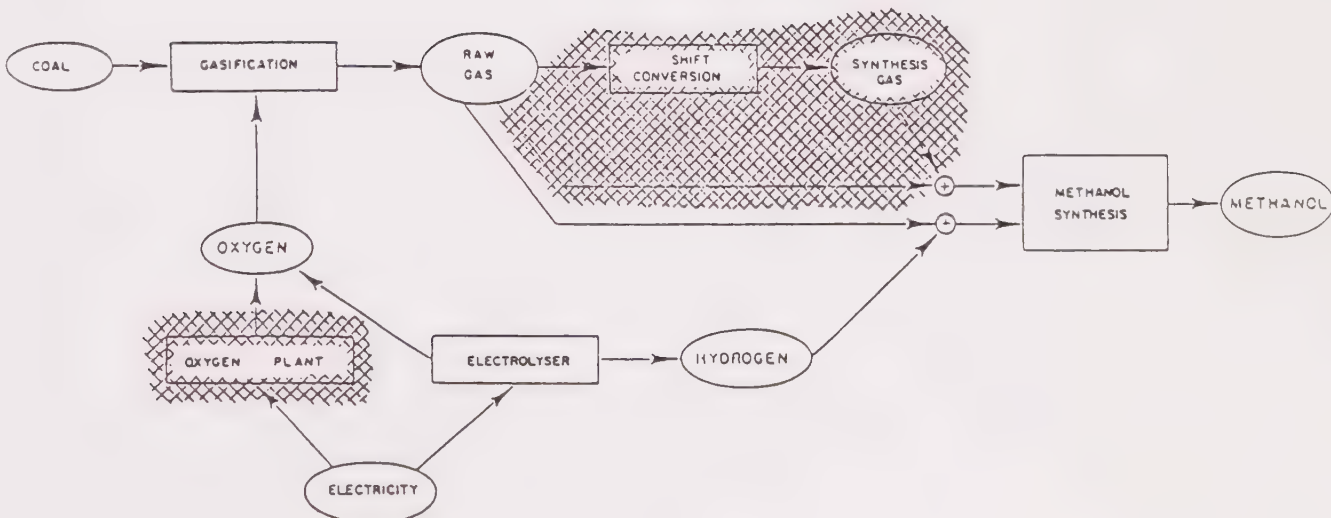


FIGURE 7: Methanol production by the coal +
electrolysis process



CONCLUSIONS

Both cycles involving methanol steam-reforming and steam injection whether including or not gas reheat can meet all the non-nuclear power requirements for periods shorter than 2000 hours per year. These new cycles can also replace decommissioned units and meet the peak power demand in the 15-25 years to come. According to economic assumptions, 10 to 20 GWe of capacity could be commissioned by the beginning of next century.

However, the outstanding efficiencies and, above all, the low construction costs of the systems examined in this preliminary study should be corroborated by further extensive studies. These studies should be carried out in close collaboration with turbine constructors who will also be responsible for making sure that these new techniques are perfectly feasible.

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SECTION II
METHANOL USE IN MOBILE SOURCES



CALIFORNIA'S METHANOL PROGRAM

EVALUATION REPORT

VOLUME I
EXECUTIVE SUMMARY



George Deukmejian,
Governor

NOVEMBER 1986

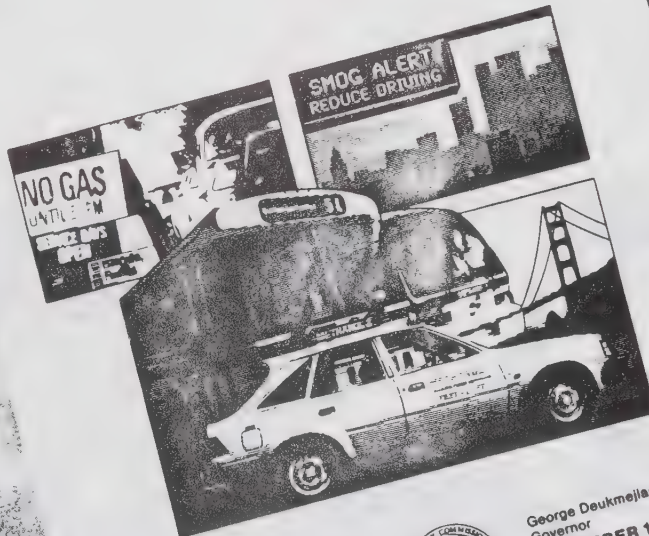
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**CALIFORNIA'S
METHANOL
PROGRAM**

**EVALUATION
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George Deukmejian,
Governor
NOVEMBER 1986
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Second Edition, November 14, 1986

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Summary

Methanol is a viable substitute for both gasoline and diesel fuels in California's motor vehicles, but barriers including costs and a fueling infrastructure have yet to be overcome.

Over the past 7 years, methanol as an alternative to gasoline and diesel fuels in California has gone from concept to reality. More than 600 light-duty, methanol-fueled vehicles have accumulated 12 million miles of day-to-day fleet service in 20 agencies around the state. At Golden Gate Transit (GGT) in San Rafael, two methanol-fueled buses have logged 84,000 miles of passenger service during the last 2 years. And in Chico, a methanol-fueled tractor was tested in farming operations at the California State University.

As a result of these programs involving a variety of representative technologies, the evidence is clear that methanol works as a fuel for passenger cars and light-duty trucks, as well as for buses and other heavy-duty equipment. While light-duty, methanol-fueled technology currently is more advanced than heavy-duty, technically there is no question that methanol is a viable substitute for both gasoline and diesel fuels.











As shown in Figure 1, the fuel economy on an energy basis of today's light- and heavy-duty, methanol-fueled vehicles is equal to the fuel economy of comparable gasoline and diesel models. In addition, it is estimated that the fuel economy for light-duty methanol vehicles can be improved 15 to 20 percent when engine designs are optimized for methanol.

Even though methanol has the potential for better fuel economy on an energy basis, one of its drawbacks is its low energy content, which is about half that of gasoline and diesel. This means that it takes about 2 gallons of methanol to go the same distance as 1 gallon of gasoline or diesel, limiting the range of methanol-fueled vehicles. Larger fuel tanks are a solution but are limited by vehicle design and size and, therefore, will not

totally compensate for methanol's lower energy content.

Methanol-fueled vehicles measure up to gasoline-fueled vehicles in performance and driveability. When used in light-duty cars and trucks, methanol's high octane number makes it a fuel that can be used in place of premium unleaded in yet higher-compression engines. When used in heavy-duty vehicles, such as tractors and methanol buses, drivers did not notice any degradation in performance or driveability.

The long-term durability of current light-duty, methanol-fueled engines looks comparable to similar gasoline-fueled engines, with many 1981 methanol-fueled Ford Escorts already accumulating over 50,000 miles. Still, to date, only three of these vehicles have

Evaluation Criteria	Worse Than Petroleum Fuels	Same As Petroleum Fuels	Better Than Petroleum Fuels
Fuel Economy		 	
Vehicle Range			
Performance		 	
Engine Durability			
Exhaust Emissions			 


 More Data Required

Figure 1. Methanol Vehicles Measure Up Well in Comparison With Their Gasoline and Diesel Counterpart Vehicles.

passed the 100,000 mile mark. The two methanol-fueled buses, which represent a much earlier stage of development than the light-duty vehicles, each have logged less than 50,000 miles. More mileage is needed on both the light- and heavy-duty vehicles to provide conclusive evidence on engine durability.

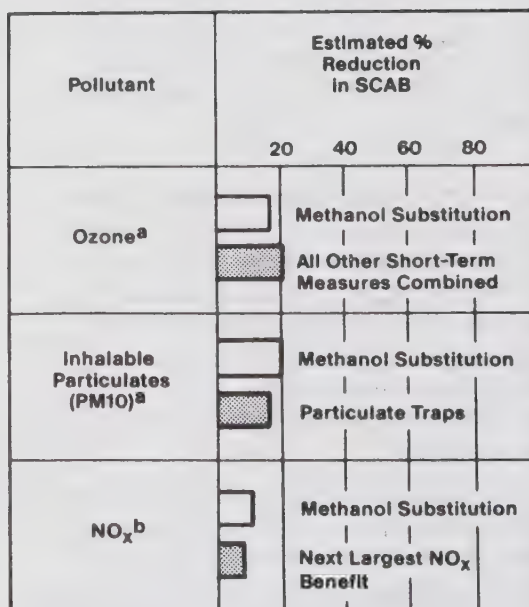
The California Methanol Program has demonstrated methanol's potential as a strategy for lowering pollution from motor vehicles. Nearly all light-duty vehicles tested easily met stringent California standards for emissions of hydrocarbons (HC), carbon monoxide (CO), and oxides of nitrogen (NO_x). In addition, 1983 methanol-fueled, electronic fuel-injection (EFI) Ford Escorts can meet the tough new California NO_x standard of 0.4 g/mile to be phased in beginning in 1989. While hydrocarbon emissions for methanol-fueled vehicles are about the same as for gasoline vehicles, methanol vehicles can substantially reduce ozone and other toxic components attributed to gasoline-fueled vehicles: hydrocarbon emissions from methanol-fueled vehicles are mostly unburned methanol molecules which are less photochemically reactive than hydrocarbon emissions from gasoline vehicles. Also, methanol emissions displace typical toxic components of gasoline exhaust, such as benzene.

Burning methanol in heavy-duty vehicles reduces NO_x emissions by 50 percent compared to diesel fuel. It also virtually eliminates fine particulates (PM10), which are inhaled deep into the lungs and may result in respiratory disease and cancer. Methanol, therefore, also is a strategy for simultaneously meeting California's strict standards for both NO_x and particulates from heavy-duty engines.

For these reasons, methanol substitution is considered an integrated control strategy for cleaner air. Substantial reductions in ozone, PM10, and NO_x are possible in the South Coast Air Basin

(SCAB) with methanol, as illustrated in Figure 2. Based on computer modeling of methanol's lower reactivity, it is estimated that if all light-duty vehicles were converted, ozone would be reduced by 20 percent. This is equal to all other short-term measures being considered by the South Coast Air Quality Management District (SCAQMD) combined. Similar reductions are projected for PM10 if methanol is substituted in heavy-duty vehicles or if an exhaust after-treatment device, called a diesel particulate trap, is developed. Methanol also offers a 10-percent reduction in total basin-wide NO_x emissions if heavy-duty vehicles are converted.

Still to be solved are problems with higher formaldehyde emissions from methanol-fueled vehicles. Work underway on improved engines and catalyst systems by manufacturers and at the Environmental Protection Agency (EPA) and the California Air Resources Board



^aReported as reduction of peak ambient levels

^bReported as reduction in tons/day of emissions

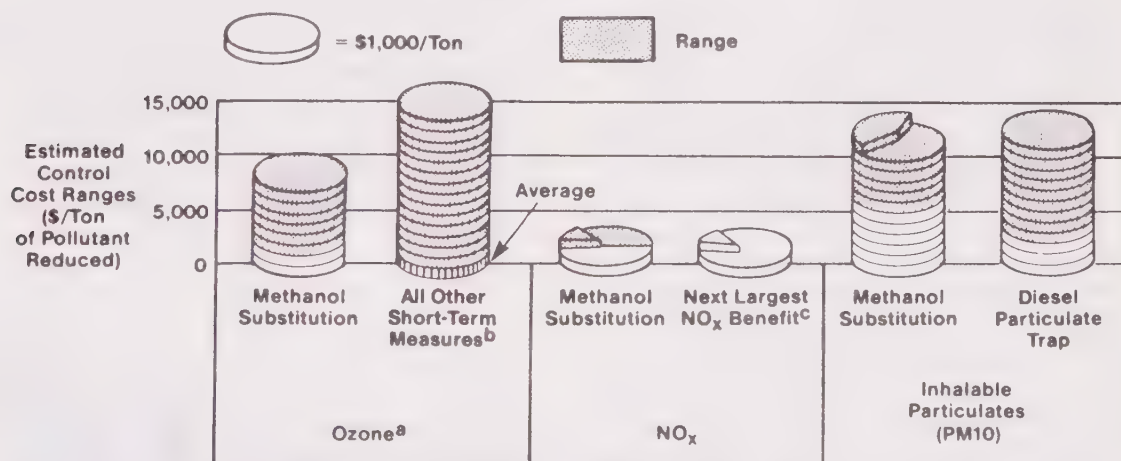
Figure 2. Methanol Substitution Offers Substantial Pollutant Reductions in the SCAB Compared With Other Proposed Strategies.

(ARB) is promising. Another unsolved problem — one which has caused frustration and inconvenience throughout the program — is the limited number of methanol fueling stations in California. Although 100 stations were planned, budget cuts and other snags have resulted in only 16 being operational, with 6 more installations in progress.

Methanol is estimated to be a cost-effective strategy for controlling pollution in California, particularly in the SCAB. As shown in Figure 3, the estimated cost per ton of pollutant reduced by methanol substitution is in the same range as other measures already in use or being considered in the SCAQMD's current Air Quality Management Plan (AQMP). Methanol's cost effectiveness depends on its price per gallon compared to that of gasoline or diesel fuels. The closer methanol's price is to about half that of gasoline or diesel, the more cost effective methanol becomes.

The methanol program has been very successful technically. Many of the problems of introducing methanol on a broad

scale have been identified and solved. It is highly recommended that the currently operating fleets be continued as a cost-effective way to obtain long-term durability data. New light-duty technology, such as improved fuel-injector systems and improved formaldehyde emission control systems, should be investigated, along with flexible-fuel vehicle prototypes. In the heavy-duty arena, improved engine designs and component reliability, along with improved emission control systems, are needed. Methanol engines need to be demonstrated in medium- and heavy-duty trucks as well. To better assess methanol substitution's effect on air quality, further analyses of the impact of methanol's lower reactivity on ozone formation are needed. Finally, transition scenarios and strategies for the large-scale introduction of methanol into the transportation fuel market need to be formulated and evaluated. A plan incorporating many of these recommendations already has been developed by the California Energy Commission (CEC), ARB, and the SCAQMD.



^aFor ozone, costs are expressed as cost /ton of "reactive" hydrocarbons (ROGs) removed from inventory.

^bLight-duty vehicle control has varied from \$400/ton of ROG for three-way catalysts to about \$1,700/ton of ROG for current I&M.

^cLight-duty NO_x vehicle control has varied from 0 to about \$1,200/ton.

Figure 3. Methanol Substitution's Estimated Cost Effectiveness is Similar to That of Other Proposed Pollutant-Control Strategies.

Background of the California Methanol Program

The California Methanol Program has shown that methanol substitution is a strategy that offers both energy security and air quality benefits.

California's Methanol Program was launched in 1979, at a time when gasoline shortages and skyrocketing oil prices had raised a public outcry about America's dependence on unpredictable energy sources. To protect California's energy security, the state legislature authorized funds "to investigate the practicality and cost effectiveness of alternative motor vehicle fuels." The CEC responded with a program to evaluate fuels capable of being domestically produced with a realistic potential as substitutes for gasoline and diesel and consistent with California's air quality goals. These alternative fuels included electricity, natural gas, methanol, ethanol, hydrogen, and propane. While it is likely that some of these fuels will find use as transportation fuels in California, methanol stood out clearly as the fuel with the best potential as a replacement for petroleum on a widespread basis.

During this time, the ARB and the SCAQMD continued their long-term assault on air pollution in the SCAB. Although great strides have been made, state and federal standards for criteria pollutants — including ozone, nitrogen dioxide, carbon monoxide, and particulates — still are being violated on more than half the days of the year, often by large percentages. Furthermore, it was becoming all too clear that if California continued with business as usual, some of these standards — particularly for ozone — would not be met in this century. This was especially evident with the number of cars, trucks, and buses in the Los Angeles area at the 7-million mark and rising. The consequences of not meeting these standards specified in the Clean Air Act include such drastic measures as cutting off highway and other federal funds to the district. Because methanol has long been known to be

cleaner burning than either gasoline or diesel, one of the objectives of the California Methanol Program became to quantify the air quality benefits of replacing petroleum fuels with methanol for motor vehicles.

Today, after 7 years of research, development, and demonstration by CEC, two things are clear: methanol *does* work as a fuel in both light- and heavy-duty vehicles; and methanol *does* offer pollution control benefits that could bring the SCAB significantly closer to clean air standards for ozone and NO₂, as well as reduce ambient levels of other toxic air contaminants. There are a number of technical details still to be worked out, but methanol substitution stands out as an integrated strategy that addresses energy security and a number of air quality problems at the same time; and, unfortunately, both energy security and air quality problems still are very real.

Although California has reduced its dependence on foreign oil since the late 1970s, long-range energy security by no means is assured. As Figure 4 shows, gasoline and diesel consumption in California continues to rise. Current low prices of oil already have begun to slow domestic petroleum production, and experts agree that increased oil use and continued reliance on foreign imports will very likely make the United States vulnerable to another oil energy crisis in the future.

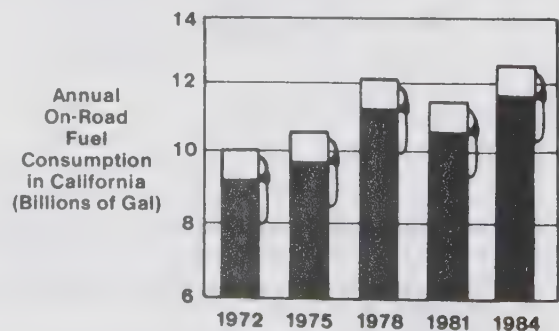


Figure 4. On-Road Fuel Consumption in California Continues to Rise.

In addition, progress in meeting air quality standards in the SCAB continues to be disappointingly slow. This is illustrated for ozone in Figure 5. New and innovative solutions are needed as badly today as ever before — particularly with the recent identification of airborne toxics, acid deposition, and inhalable particulates as serious health hazards in urban areas such as Los Angeles.

Whether or not methanol substitution will be used as a strategy for energy security and air quality in California has yet to be decided. This report on CEC's work during the past 7 years provides the facts and figures needed to make an informed decision.

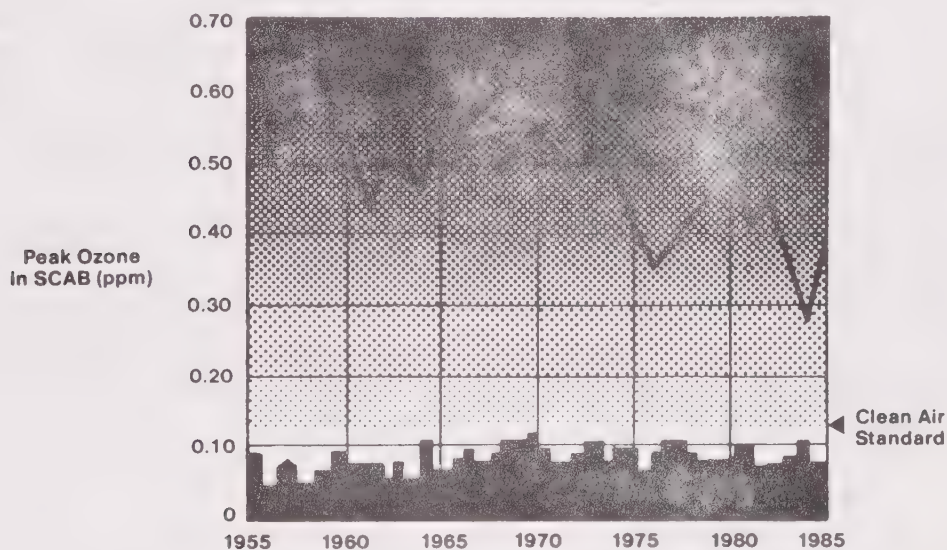


Figure 5. While Much Progress Has Been Made in Reducing Ozone in the SCAB, Peak Levels Still Are Well Above Healthful Standards.

The Light-Duty (Automobile) Vehicle Program

Methanol-fueled, light-duty vehicles compare favorably to gasoline-fueled models in energy fuel economy, performance, and most pollutant emissions. Long-term engine durability, formaldehyde emissions control, and range and fueling convenience are questions that remain to be answered.

The California Methanol Program has made a significant contribution to the advancement of methanol-fueled, light-duty vehicle technology. Since 1980, 600 methanol- and ethanol-fueled vehicles in 4 fleet demonstrations have logged more than 12-million miles on the road. Over 500 methanol-fueled Ford Escorts currently are being used in day-to-day service by 20 state and local agencies and 2 private fleets all over California. This is the largest fleet of methanol-fueled vehicles ever to be tested anywhere in the world and is continuing to accumulate operating experience at a rate of 4 million miles per year.

Because methanol is more corrosive than gasoline to some metals and plastics used in engines and fuel systems, gathering accurate data on long-term engine durability has been a major objective of CEC's demonstration programs. While engine wear was, in fact, a problem in the early stages of the program, fleets of 1981 and 1983 methanol-fueled Ford Escorts have shown little or no evidence of abnormal engine wear, even in a few vehicles that have logged over 100,000 miles. Continuing this program until a large number of these Escorts are driven in excess of 100,000 miles is critical to establishing conclusive, long-term durability results for light-duty, methanol-fueled vehicles (Figure 6).

Methanol-fueled vehicles in all four demonstration fleets were also compared to gasoline-fueled controls in terms of fuel economy. Methanol-fueled engines achieved better mileage on an energy

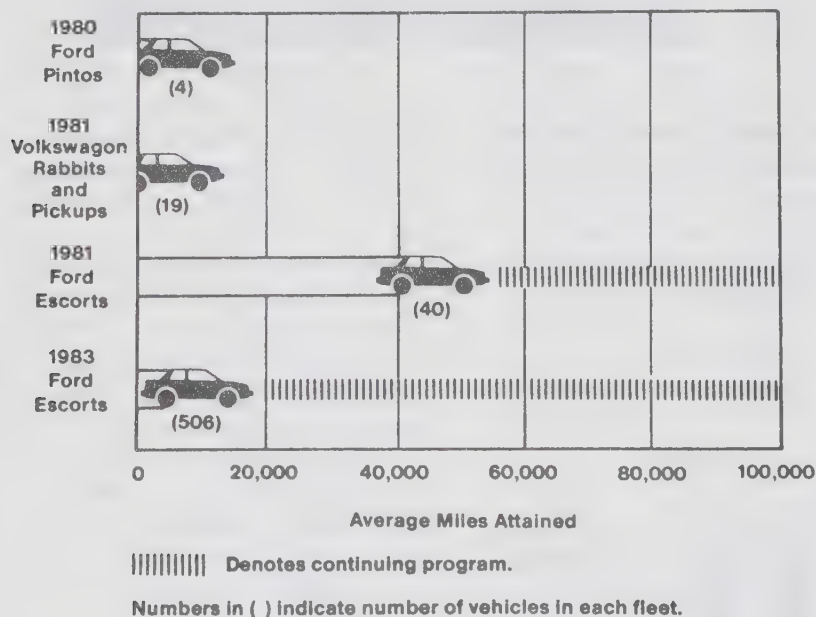


Figure 6. Much More Mileage Needs to be Accumulated on the 1983 Ford Escorts to Provide Firm Conclusions on Engine Durability.

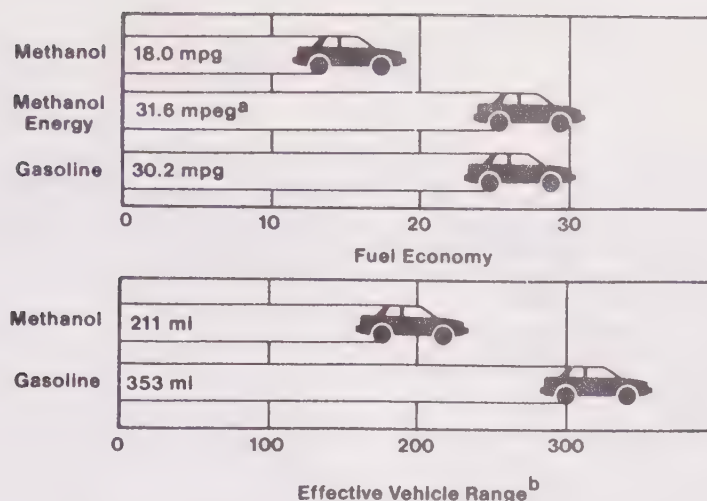


Figure 7. Methanol 1983 EFI Ford Escorts Achieved Better Fuel Efficiency on an Energy-Equivalent Basis Than EPA Certification Data for Similar Gasoline-Fueled Escorts.

equivalent basis* than control vehicles or EPA certification data in all but one program. Typical data is shown for the 1983 EFI Ford Escorts in Figure 7. In addition, although these engines were not fully optimized for running on methanol, vehicles in the last three demonstration fleets have met and surpassed 1985 Corporate Average Fuel Economy (CAFE) standards.

When asked to rate methanol-fueled vehicles for driveability and performance, drivers in all four fleets gave high marks for acceleration and response when the vehicles were operating properly. This rating was borne out by CHP acceleration tests in which 1983 methanol-fueled Ford Escorts outran a similar 1984 gasoline-fueled model by almost 4 seconds from 0 to 60 mph. However, both methanol-related problems (carburetor float deterioration and vapor lock), as well as nonmethanol-related problems (transmission failure and power steering malfunction) have resulted in mixed reviews in terms of performance for methanol-fueled vehicles.

The most serious problems during the four demonstration programs have been encountered not with the vehicles themselves but with the methanol fueling stations. Although a network of 100 stations throughout California was originally planned, budget cuts and other difficulties have limited the number of stations currently in service in the entire state to 16, with 6 more underway (Figure 8). This has made drivers reluctant to use methanol-fueled vehicles — especially on longer trips, since methanol-fueled vehicles require refueling more often due to their limited range. In addition, the cardlock system used to control fuel access and handle billing at the stations has proven unreliable in some instances, causing drivers further frustration.

Short of a regulatory policy, the best answer to the "chicken and egg" problem of methanol-fueled vehicles and the fueling stations needed to support them may be flexible fueled vehicles (FFVs). FFVs, now being tested by Ford, are equipped with an electronic control system that makes it possible to run on methanol, gasoline, or any mixture of the two from a single fuel tank. Until the time that there are enough methanol-fueled vehicles on the road to warrant a widespread

*Methanol has about half the energy content of gasoline or diesel: it takes about 2 gallons of methanol to provide the energy of 1 gallon of gasoline or diesel.

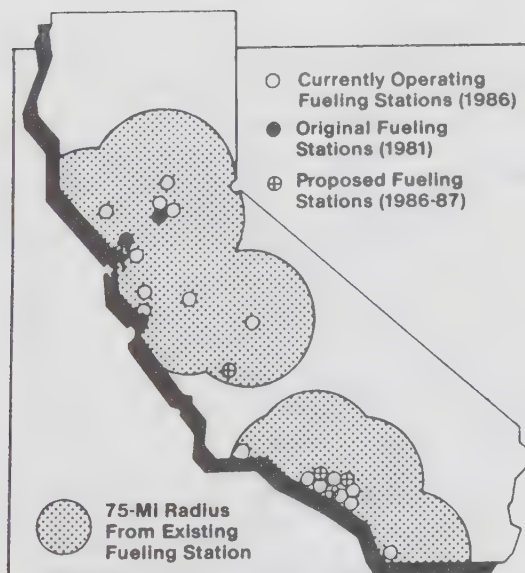


Figure 8. Poor Methanol Fueling Station Density Continues to Hamper Fleet Vehicle Use, Despite the Construction of New Facilities in 1986 and 1987.

network of fueling stations, FFVs could eliminate some of the difficulties the California Methanol Program has encountered.

Along with durability, fuel economy, and performance, vehicles in all four demonstration fleets also were tested for exhaust and evaporative emissions. These tests have demonstrated that methanol-fueled engines can meet both

current and more stringent California emissions standards for hydrocarbons, carbon monoxide, and oxides of nitrogen. For the 1981 and 1983 Escorts, these emission levels — as well as acceptable levels of evaporative emissions — were achieved by engines using M85*. The recent development by Toyota of an advanced engine with electronic fuel injection and an oxygen sensor for lean-burn control promises improved fuel economy using M85 without increasing emissions.

One final problem with methanol fueling in light-duty vehicles which has not yet been solved is the failure of existing emission control systems (designed for gasoline) to reduce formaldehyde emissions. Except for Toyota's advanced lean-burn engine, formaldehyde emissions from most methanol-fueled vehicles tested have been significantly higher than those from similar gasoline-fueled models. Work underway at EPA and ARB indicates that exhaust catalyst systems can be developed to reduce formaldehyde emissions from methanol-fueled engines to the level of current gasoline engines. Developing and testing more effective engine and catalyst systems thus is essential for taking full advantage of methanol's air quality benefits.

*M85 is a mixture of 15-percent unleaded premium gasoline and 85-percent neat methanol. Gasoline is added as an aid to cold starting in light-duty vehicles and to increase safety; bus programs are using neat methanol.

The Heavy-Duty (Truck and Bus) Vehicle Program

In spite of being a new technology, methanol-fueled, heavy-duty engines can match the performance and energy fuel economy of diesel engines, plus offer a strategy for meeting stringent new emissions regulations.

Compared to the light-duty sector, heavy-duty, methanol-fueled engine technology is fairly new. The world's first methanol-fueled bus was put into revenue service late in 1980 in Auckland, New Zealand. Demonstration programs in West Germany and South Africa followed in 1981 and 1982. When the CEC and GGT took delivery of the two methanol-fueled buses built by General Motors (GM) and M.A.N. of West Germany, they were the fifth and sixth in the world and the first to be tested in revenue service in the United States. Another heavy-duty methanol vehicle program involves a methanol-fueled tractor supplied by Komatsu of Japan, which has been tested in farming operations for over 1,400 hours of service at the California State University at Chico.

The first methanol coach delivered to GGT in August 1983 was built by GM. The engine, a methanol-fueled version of the most widely used two-stroke diesel bus engine in the United States, was the first ever built for compression ignition with 100-percent methanol. Because it was delivered after only 2 years of research and development in Detroit, there were initial difficulties, including poor driveability, fuel-injector failures, and methanol compatibility problems. Working out these technical details took the better part of a year and resulted along the way in an embarrassing nonmethanol-related engine failure caused by a burned valve at the televised press conference to introduce the program.

Because M.A.N. of West Germany has been developing methanol-fueled engines for nearly 15 years, the introduction of its coach was much smoother.

M.A.N. also supplied a standard diesel coach as a control vehicle. Since the M.A.N. engine is a four-stroke, spark-ignited model, CEC has been able to evaluate the advantages and disadvantages of two different engine designs advanced by M.A.N. and GM.

Once the initial technical problems were solved, both coaches were placed in regular passenger service. As shown in Figure 9, the GM coach has logged over 37,000 miles, while the M.A.N. bus has accumulated nearly 50,000 miles of service.

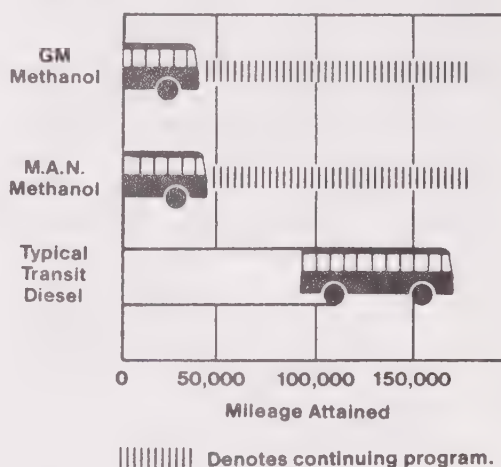


Figure 9. Much More Mileage Needs to be Accumulated on Both Buses to Form Conclusions on Engine Durability.

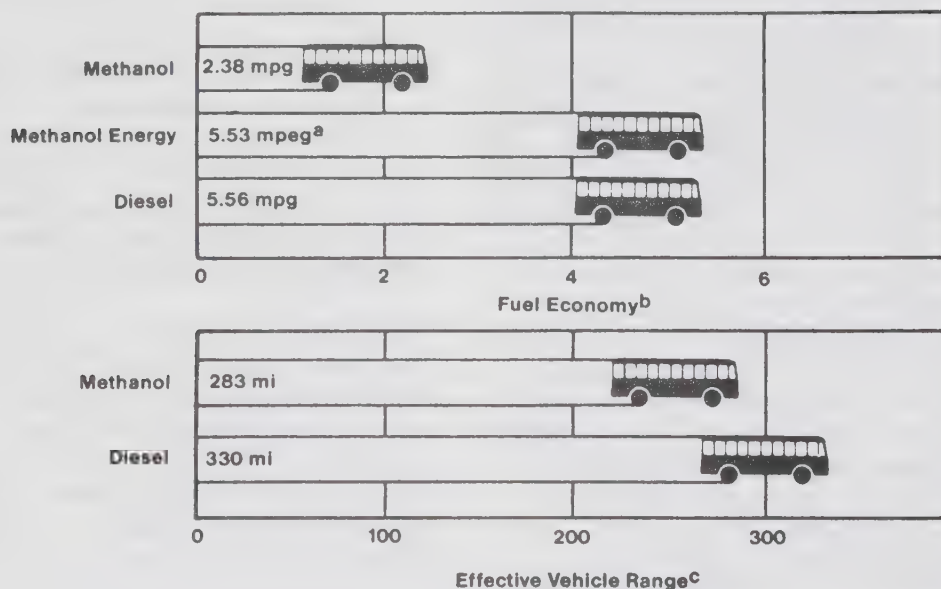
Until the GM and M.A.N. coaches log a minimum of 10,000 hours, or 100,000 miles of operation, no firm conclusions can be drawn about the long-term durability of methanol-fueled, heavy-duty engines. Preliminary results, however, indicate that problems with GM's fuel pump and fuel injectors and with M.A.N.'s spark plugs and catalyst system are solvable technical problems. These are typical types of problems which occur when introducing new technologies. While the GM engine needs more work at this point than the M.A.N., both designs appear to have the potential for satisfactory long-term service.

Both buses were tested for fuel economy and compared to diesels. As shown in Figure 10, in both road tests and actual revenue service, the M.A.N. methanol-fueled engines' fuel economy is almost identical to that of a similar diesel on an energy equivalent basis. The less-developed GM methanol-fueled engines' fuel economy is about 20 percent less than that of a similar diesel. For buses, fuel tank capacity can be expanded to give a vehicle range comparable to that of a diesel.

Both methanol-fueled engines have been extensively tested for exhaust emissions. These tests indicate that future methanol-fueled, heavy-duty engines can meet or exceed tougher 1991 emission standards for NO_x and particulates. The current GM and M.A.N. engines both are prototypes and are not fully optimized for emission control. In fact, testing of the GM engine showed NO_x 50 percent

lower than the 1991 standard. Particulates were reduced by over 350 percent but still are higher than the 1991 standard. More engine development is required in order to achieve this standard. Similar testing of the M.A.N. engine showed NO_x equal to the 1991 standard and virtually no particulate. Expected improvements to these engines will better their performance, making them a viable strategy for meeting more stringent California standards.

In contrast to CEC's light-duty fleet demonstrations, fueling the buses has not been a problem. Fuel tanks are filled each day at a central location and, since the buses operate over a set route, vehicle range has not been a drawback. As in the light-duty fleet programs where several methanol fuel mixtures have been used, no safety problems have been encountered with neat methanol in the heavy-duty programs.



^aBased on 2.38 mpg using M100 and energy equivalence ratio of 2.32

^bFuel economy for M.A.N. buses

^cAssuming 66-gal fuel tank for gasoline, 132-gal for methanol with 10% reserve

Figure 10. The M.A.N. Methanol Bus Has Achieved Equal Fuel Economy to its Diesel Counterpart on an Energy-Equivalent Basis.

Once initial difficulties were worked out, drivers rated the performance and driveability of both methanol-fueled buses as equal to that of diesels. As shown in Figure 11, acceleration test results confirmed the drivers' experience. Questionnaires filled out by passengers also showed no perceived difference in performance between the methanol-fueled buses and other coaches the respondents had ridden.

Because buses are major contributors to street-level pollution in cities, continuing CEC's bus demonstration project to work out technical problems and establish the long-term durability of methanol-fueled engines is important. Increasing the number of buses tested also will be important so that the methanol coaches are fueled and maintained like the rest of the diesel fleet and not handled separately as in the current GGT demonstration. Further, it is important to introduce methanol buses to different transit districts to help familiarize the California transit industry with this technology.

Buses represent a small percentage of the diesels on the road compared to medium- and heavy-duty trucks. In addition, because truck operating conditions are different from those of buses, suc-

cessful demonstrations of methanol engines in buses do not assure that the technology can be transferred. Future demonstrations of medium- and heavy-duty trucks are needed to broaden the application of methanol engines. Substituting methanol in these truck sectors will substantially reduce emissions of NO_x and particulates in California.

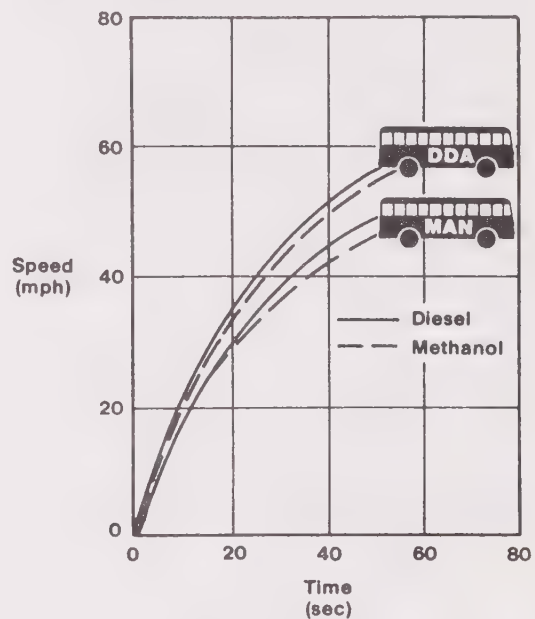


Figure 11. Methanol and Diesel Coaches Have Similar Acceleration Performance.

Methanol and Air Quality

Substituting methanol for gasoline and diesel fuels is an integrated strategy with the potential for simultaneously reducing emissions of a number of major air pollutants by substantial amounts.

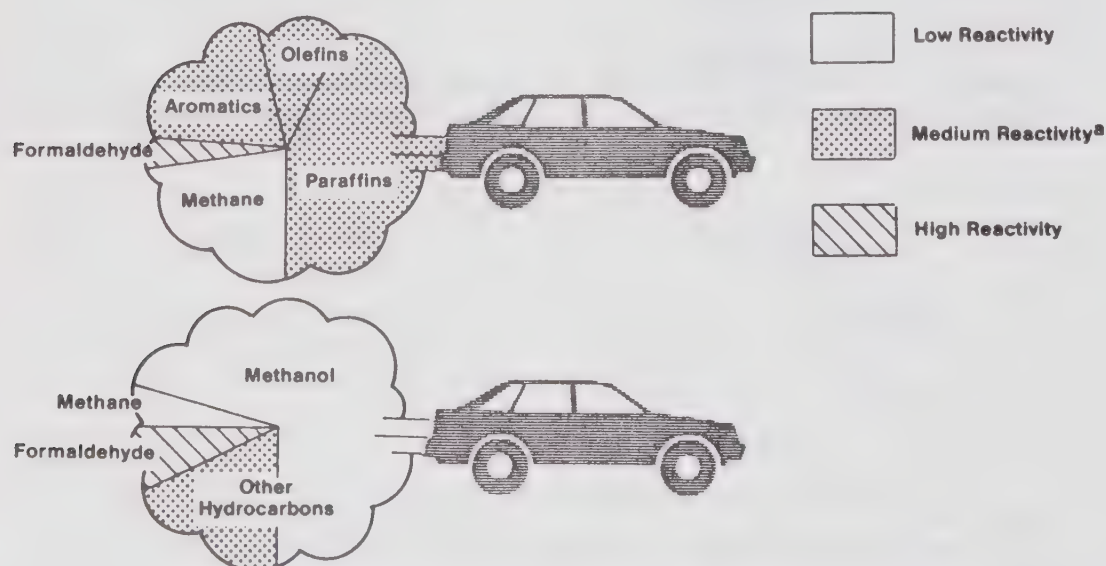
By far the most severe air pollution problem facing the SCAB is ozone. Although progress has been made in reducing ozone pollution, ambient ozone levels continue to violate federal air quality standards by substantial amounts in the Los Angeles area.

Ozone is not emitted directly but is formed in the atmosphere when hydrocarbons and oxides of nitrogen (NO_x), called ozone precursors, are acted on by sunlight. Strategies for reducing ozone in the SCAB have focused on lowering emissions of these precursors — particularly reactive hydrocarbons — from light-duty vehicles as well as oil refineries, power plants, gasoline stations, and other sources.

As a result of these control measures, hydrocarbon emissions have been reduced from 1,950 tons per day in 1966 to

about 1,400 tons per day in 1983. However, to meet the federal standard for ozone, the SCAQMD estimates that hydrocarbon emissions will need to be lowered to less than 300 tons per day — a reduction of almost 1,100 tons per day. The 1982 SCAQMD AQMP can identify only about 200 additional tons of hydrocarbons that can be eliminated by the year 2000 without imposing strict control measures, such as severely restricting private automobile travel and urban growth. New solutions are clearly called for.

Substituting methanol for gasoline in light-duty vehicles appears to offer such a solution. As shown in Figure 12, the exhaust from methanol-fueled engines contains mostly unburned methanol, a hydrocarbon that reacts much more slowly to form ozone than the hydrocarbons in gasoline exhaust. Because of this property, air quality computer models have predicted that methanol substitution has the potential for bringing about a greater reduction in ozone than all other hydrocarbon control strategies in



^aIndividual chemical species in these groups have range of reactivity from low to high.

Figure 12. Hydrocarbons in the Exhaust of Methanol-Fueled Vehicles Are Composed Mainly of Unburned Methanol, Compared to the More Reactive Hydrocarbons in Gasoline Exhaust.

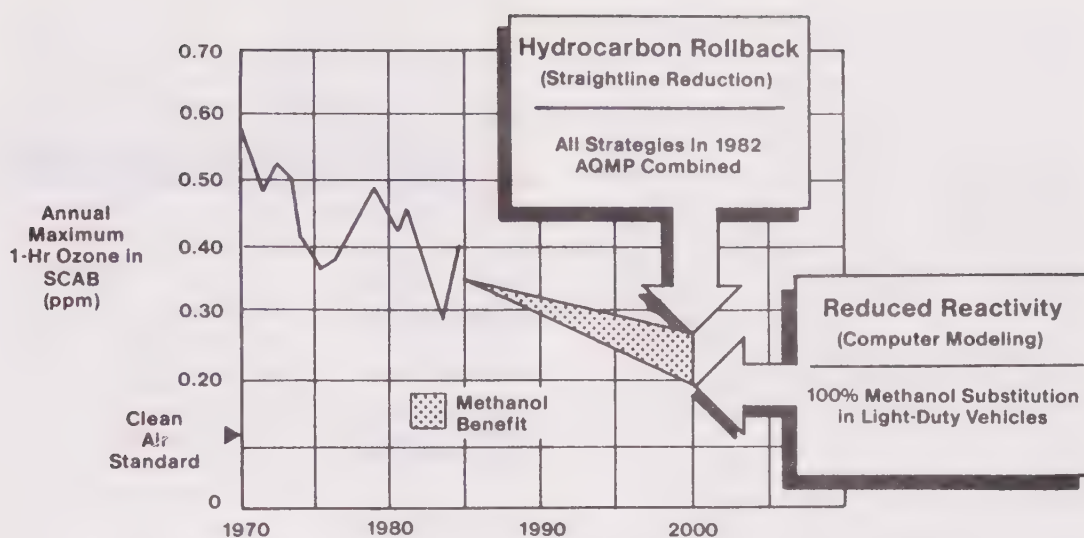


Figure 13. Substituting Methanol in Light-Duty Vehicles in the SCAB Could Reduce Peak Ozone Levels by the Year 2000, More Than All Other Strategies in the 1982 South Coast Air Quality Management Plan.

the 1982 South Coast AQMP combined. If these predictions are accurate, implementing methanol substitution, along with the other hydrocarbon control measures in the South Coast AQMP, could bring the SCAB within striking distance of meeting the federal ozone standard, as Figure 13 shows.

Both current smog chamber research and air quality modeling indicate that methanol substitution offers significant benefits on the first day of a multiday air pollution episode like those common in the Los Angeles area in the summer. The benefits on the second and third day of such episodes are less well understood, although they are still believed to be substantial. Research is underway to better quantify these benefits.

The accuracy of the few modeling studies performed on methanol substitution is also uncertain. While the computer models used for nearly two decades to analyze ozone control strategies are adequate, better modeling of formaldehyde and nighttime reactions — along with sensitivity analyses — are needed to accurately quantify the benefits of methanol substitution in the

SCAB. The ARB recently issued a contract for updated computer studies to determine these benefits.

With more accurate modeling, the benefits of methanol substitution can be more accurately assessed, and research can be focused on technical problems that remain unsolved. The alternatives facing the SCAB — government restrictions on automobile use and urban expansion — make exploring methanol's potential as an ozone control strategy an important task to accomplish.

For heavy-duty diesel engines, the EPA and California ARB recently promulgated stringent new emissions standards to control NO_x and particulates. Under these standards, current diesel engines will not be in compliance without the addition of a trap to eliminate particulates from the exhaust. Significant improvements in diesel fuel quality will likely be necessary to ensure the effectiveness and durability of such traps. Lower NO_x emissions from diesels will be achieved with recently introduced electronic controls. As an alternative strategy to traps and cleaner diesel fuel, methanol substitution has the potential

to simultaneously cut NO_x emissions in half and virtually eliminate particulates (see Figures 14 and 15).

Oxides of nitrogen continue to be a pollution problem in the SCAB, although attainment of the ambient nitrogen dioxide (NO_2) standard is projected for 1988. Besides playing a role in the formation of ozone, NO_x causes brown haze, reduces visibility, and is a major contributor to acid deposition. Over 1,100 tons of NO_x per day are emitted into the atmosphere in the SCAB, roughly 60 percent from motor vehicles. Attainment is being projected because of California's stringent emission regulations on light-duty vehicles and stationary sources. With current three-way exhaust catalysts, NO_x emissions from methanol- and gasoline-fueled, light-duty vehicles are roughly comparable.

Because of its lower combustion temperature and capability for lean burning, heavy-duty engines using 100-percent methanol emit only about half as much

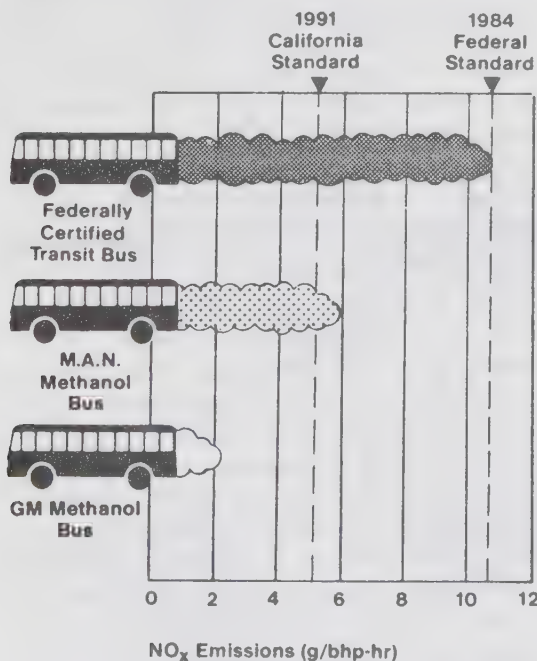


Figure 14. Methanol Substitution in Heavy-Duty Vehicles Reduces NO_x Up to 50 Percent.

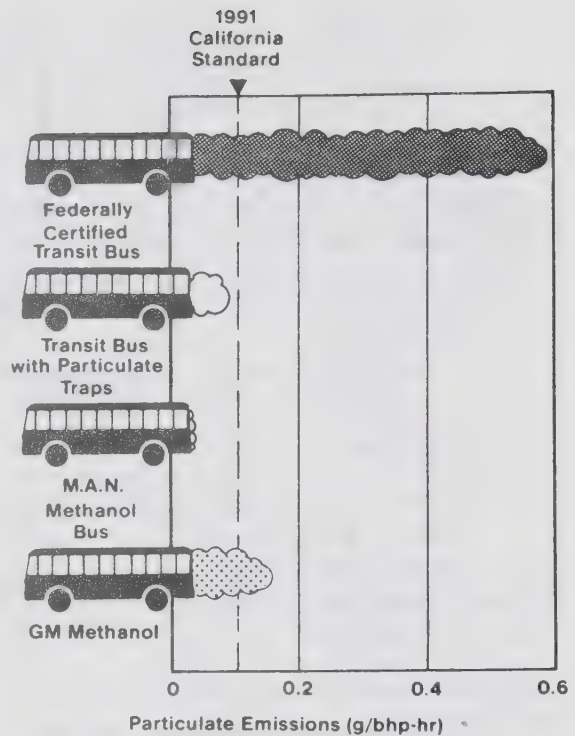


Figure 15. Methanol Substitution in Heavy-Duty Vehicles Can Virtually Eliminate Particulate Emissions.

NO_x as current diesels, as shown in Figure 14. As a result, substituting methanol for diesel fuel in heavy-duty vehicles could be a major step toward lowering ozone levels and keeping the SCAB in attainment for NO_2 .

At the same time, because methanol engines emit virtually no particulates, substituting methanol for diesel in heavy-duty engines would meet the tough new particulate standard, as shown in Figure 15. Meeting this standard by either methanol or traps would improve visibility in the Los Angeles area by an estimated 25 to 30 percent. More importantly, emissions of fine particulates (PM_{10}) — known to cause respiratory disease and suspected of contributing to cancer — would be significantly reduced, as shown in Figure 16. This would remove a significant health hazard from our urban environment.

Methanol substitution in both light- and heavy-duty vehicles is also a potential strategy for greatly reducing emissions of toxic or carcinogenic substances like benzene, benzo-(a)-pyrene, xylene, and toluene, which are found in gasoline and diesel exhaust but are not produced by methanol combustion.

However, formaldehyde — another toxic and suspected carcinogen, as well as a contributor to ozone formation — has been measured in higher concentrations in methanol exhaust than in the

exhaust from gasoline and diesel engines. Exceptions have been the advanced lean-burn engine from Toyota, which has formaldehyde emissions comparable to gasoline vehicles, and the M.A.N. bus engine, which uses a catalyst to control formaldehyde emissions to levels lower than from diesel engines. Further development work and research is necessary to continue improving formaldehyde control. Both EPA and ARB currently are pursuing this research.

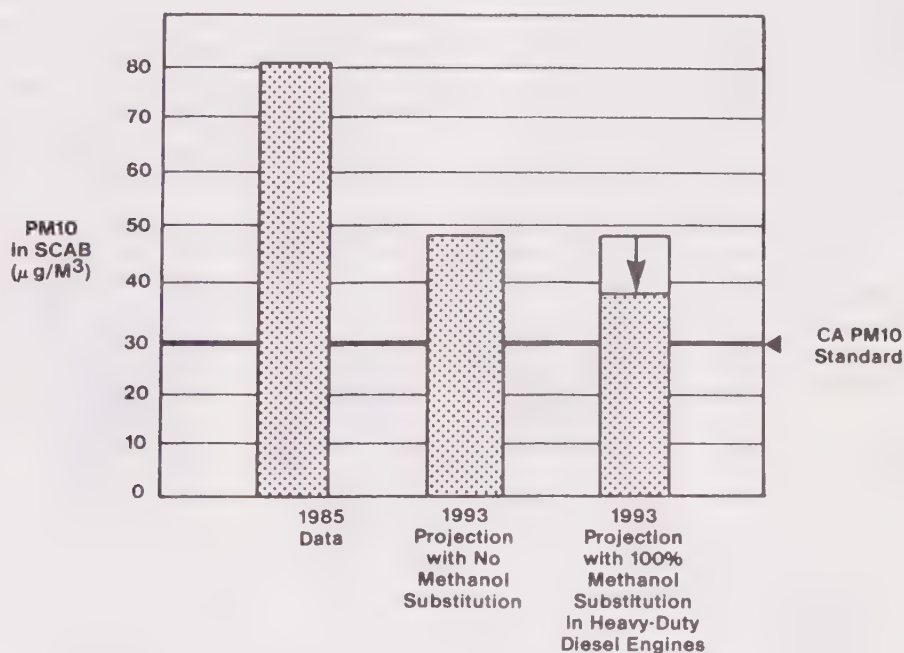


Figure 16. Methanol Substitution in Heavy-Duty Vehicles Would Significantly Reduce Inhalable Particulate Levels (PM10) in the SCAB.

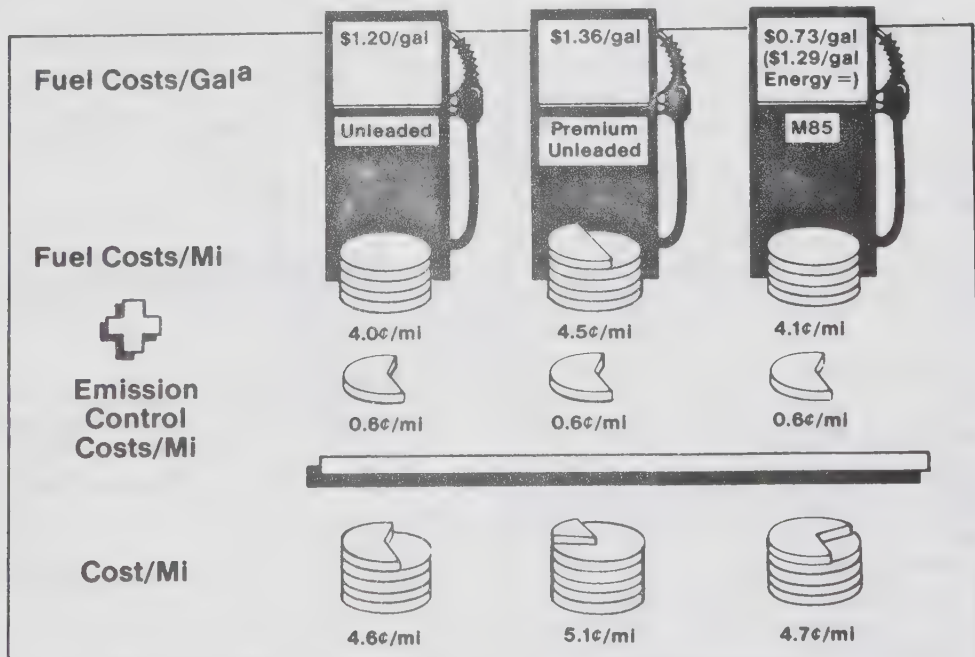
Cost Effectiveness of Methanol for Ozone Control

The cost of methanol substitution as an ozone control strategy is in the same range as other strategies proposed in the 1982 South Coast AQMP.

Substituting methanol for gasoline in light-duty vehicles as a strategy for controlling ozone pollution has many parallels to the shift from leaded to unleaded gasoline throughout the United States. As with unleaded gasoline, methanol can be a cost-effective strategy only if it is applied on a widespread basis to bring down the cost of producing and distributing methanol through economies of scale. Also, like the differences between cars that burn leaded and unleaded gasoline, the differences between gasoline-fueled and methanol-fueled vehicles are minor. Mass-produced, a methanol-fueled car or light-duty truck will not cost any more than a comparable

gasoline-fueled model. As with regular compared to unleaded gasoline, the price of methanol substitution will show up mainly in the price at the pump. Finally, methanol is like unleaded in being an integrated control strategy that offers emissions control benefits for several different pollutants, including reactive hydrocarbons and airborne toxics from light-duty vehicles and oxides of nitrogen (NO_x), particulates, and airborne toxics from heavy-duty vehicles.

Unleaded gasoline plus the catalytic emission control systems and smog certification programs that have come with it have added about 0.9 cents per mile or about \$100 per year to the average cost of operating a vehicle in California. Depending on the relative prices of oil and methanol, it is estimated that substituting methanol for gasoline would



^aSource of gasoline fuel prices: Lundberg Survey, Inc.

Assumptions:

- 1985 Fuel Prices
- Low Transportation Costs
- Includes Dealer Markup, Road Tax, and California Sales Tax

Figure 17. Methanol is Price Competitive With Both Regular and Premium Unleaded Gasoline on a Cents-Per-Mile Basis.

add an additional 0.1 cent per mile, or about \$10 per year, to this cost. As shown in Figure 17, the pump price of methanol is higher than the price of regular unleaded, but below the price of premium unleaded. Since methanol is a premium fuel (octane number 102 compared to 91 for premium unleaded and 87 for regular unleaded), drivers that now purchase premium actually would save money with an optimized methanol-fueled vehicle. Drivers who normally would buy regular unleaded would pay a small penalty — though only about half the penalty of switching from regular gasoline to regular unleaded.

As a strategy for reducing ozone in the SCAB, methanol substitution in light-duty vehicles offers relatively little advantage over gasoline for NO_x , but a substantial benefit in terms of reactive hydrocarbons. Assuming that unburned methanol in vehicle exhaust is unreactive, substituting methanol for gasoline in light-duty vehicles could yield a reduction in reactive hydrocarbons equivalent to 170 tons per day — an amount nearly

equivalent to all other hydrocarbon reduction strategies proposed in the SCAQMD's 1982 AQMP. The cost of this strategy would be about \$2,200 per ton of reactive hydrocarbons displaced.

As shown in Figure 18, this is higher than the cost of current strategies, like exhaust catalysts and vehicle inspection and maintenance programs. But it is much less than the cost of some of the other hydrocarbon reduction strategies in the 1982 South Coast AQMP, such as carbon absorption systems for rubber products plants, which have costs as high as \$15,000 per ton. Furthermore, the costs for other strategies that will yield reductions in reactive hydrocarbons comparable to those offered by methanol substitution — rearranging urban layout, removing petroleum refining from SCAB, increasing mass transit, and possibly limiting industrial growth — have not yet been calculated or even estimated.

Methanol substitution thus appears to be a potentially cost-effective ozone control strategy for the SCAB.

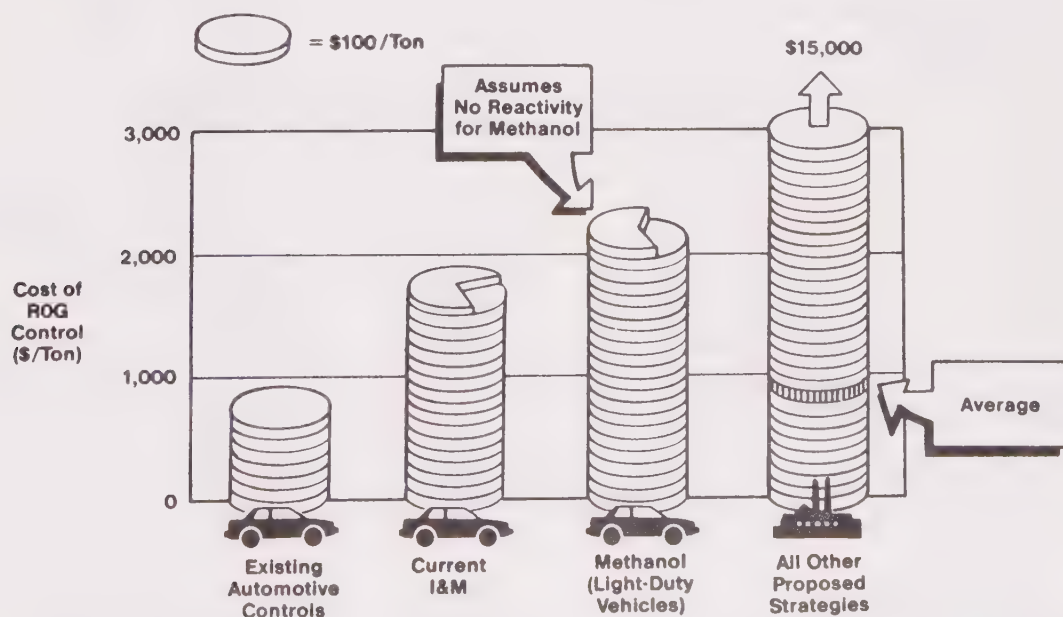


Figure 18. Methanol's Cost Effectiveness as a Hydrocarbon Control Strategy is Higher Than the Cost of Current Strategies, But Much Less Than the Cost of Some Proposed Strategies.

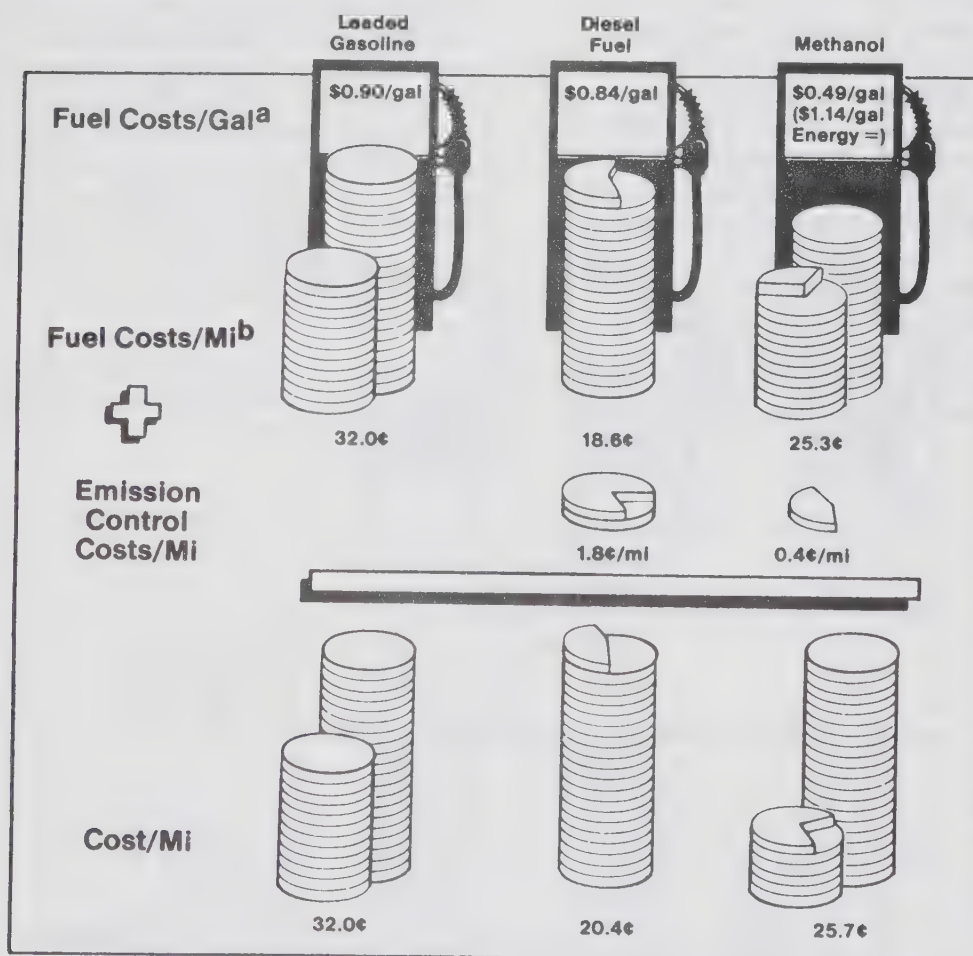
Cost Effectiveness of Methanol for NO_x and Particulate Control

The cost of methanol substitution as a strategy for controlling NO_x and particulates from heavy-duty vehicles is in the same range as other vehicle control strategies and much lower than costs for controlling these pollutants from stationary sources.

In terms of price at the pump, methanol does not compare as well to diesel fuel as it does to unleaded regular and unleaded premium gasoline. As shown in Figure 19, diesel, which is a less refined product than gasoline, is substan-

tially cheaper than methanol. In addition, although methanol-fueled, heavy-duty engines are about 50 percent more efficient than gasoline-fueled engines, methanol and diesel engines have about the same efficiencies. Thus, before figuring in the cost of emission controls, the cost per mile of methanol for transit bus operation is roughly 25 cents compared to diesel's cost per mile of about 19 cents.

Heavy-duty trucks and buses, however, are facing much more stringent



^aSource of gasoline and diesel fuel prices: Lundberg Survey, Inc.

^bTypical transit bus application

Assumptions:

- 1985 Fuel Prices
- Low Transportation Costs for Fuel
- Does Not Include Dealer Markup or Road Tax

Figure 19. Methanol is More Expensive Than Diesel But Less Expensive Than Gasoline for Heavy-Duty Vehicles on a Cents-Per-Mile Basis.

new emissions standards for both particulates and NO_x in the next few years. Particulates, which are now unregulated and about 0.6 g/bhp-hr, will be controlled to 0.1 g/bhp-hr for buses in 1991. Heavy-duty trucks will be required to meet this tough standard in 1994. Federal NO_x emissions standards for these engines also will be lowered to 5.0 g/bhp-hr in 1991 — similar to California's current standard for heavy-duty engines.

Because methanol-fueled, heavy-duty engines emit only about 50 percent as much NO_x and virtually no particulate matter compared to diesels, methanol substitution is a potential strategy for meeting the new emissions standards for both of these pollutants simultaneously. For NO_x, diesel engine manufacturers plan to comply with tougher emissions standards with electronic engine controls. The cost of this strategy is expected to be very low. Figure 20 compares the cost of methanol substitution to

existing California NO_x control strategies for heavy-duty and light-duty vehicles. Also given for comparison is the range of NO_x control costs for typical stationary sources in the SCAB. As shown, the cost of methanol substitution is comparable to current vehicle control strategies and considerably cheaper than costs incurred for controlling stationary sources with advanced technologies.

For meeting more stringent particulate emissions standards, the major strategy being considered by diesel manufacturers is particulate traps. A reliable and effective particulate trap has not been demonstrated to date. Studies predict, however, that a trap with an efficiency of about 85 percent and a life of 100,000 miles can be built for a cost of between \$1,000 and \$4,000. As shown in Figure 21, such a trap would add about 2.2 cents per mile to the operating costs of a diesel truck or bus, which translates to a cost of

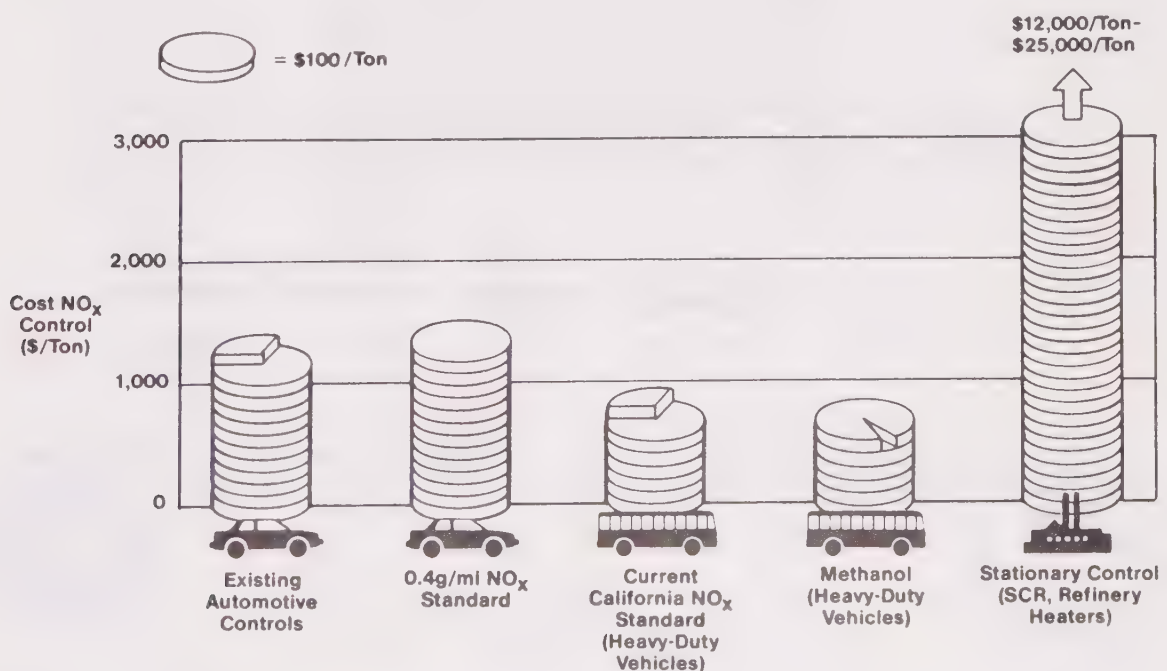


Figure 20. Methanol's Cost Effectiveness as a NO_x Control Strategy Compares Favorably With That of Other Current and Proposed Strategies.

about \$4,000 per ton of particulates eliminated. If, as some experts predict, a cleaner diesel fuel is required along with a trap to meet the new emissions standards, the cost increases dramatically to about \$12,000 per ton. The actual costs of particulate traps, once developed, will probably fall between these two costs. The cost of methanol substitution is estimated at \$6,000 per ton with the fuel price difference shown in Figure 19. The cost effectiveness of these particulate control options is shown in Figure 21. Also shown for comparison is the estimated cost if gasoline were once again used in heavy-duty engines. As diesel becomes cheaper compared to methanol, the cost of methanol substitution as a control strategy goes up and, conversely, comes down as the price difference widens.

Methanol substitution is attractive because it deals with emissions of NO_x and particulates at the same time, whereas in diesel engines if NO_x is lower, particulates increase and vice-versa. Substituting methanol for diesel also will lower CO emissions in the SCAB, as well as eliminate a number of airborne toxic and carcinogenic compounds in diesel exhaust. While methanol will probably be about 10 percent more effective at removing particulates than traps, they both will reduce fine particulate emissions in the SCAB by an estimated 20 tons per day, improving visibility and reducing the risk of respiratory disease and lung cancer.

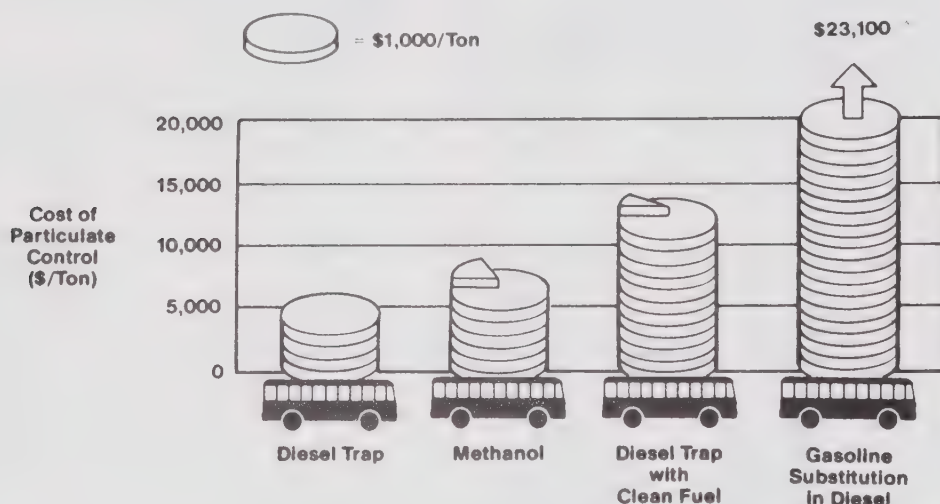


Figure 21. Methanol's Cost Effectiveness as a Particulate Control Strategy is Higher Than That for Diesel Traps, But Lower if Cleaner Fuel is Required With the Traps.

Report of the Three-Agency Methanol Task Force

Volume I — Executive Summary

May 15, 1986



Air Resources Board



South Coast Air Quality Management District



California Energy Commission

P500-86-011

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BACKGROUND

On June 29, 1984, Air Resources Board (ARB) members Roberta Hugan and Tom Hamilton, South Coast Air Quality Management District (SCAQMD) members Tom Heinsheimer and Larry Berg, and California Energy Commission (CEC) Commissioners Charles Imbrecht and Geoffrey Commons chaired a symposium on the use of methanol as an alternative fuel in California's South Coast Air Basin, as well as in other areas of the state.

At the conclusion of the symposium, the panel directed the staff of the three agencies to establish a joint Task Force to develop an action plan to guide each agency's involvement in the evaluation and commercialization of methanol as a fuel in California. The combined three-agency Task Force was directed to assess the full range of issues affecting the use of methanol in both mobile and stationary applications. The Task Force was also charged with developing an action plan for further research, technology development, and commercialization demonstration projects which would be consistent with local, state, and federal air quality strategies. This report describes the results of the cooperative interagency effort to develop that action plan.

In developing the action plan and the specific projects for carrying it out, improved air quality has been given the highest priority. Because of the Los Angeles area's severe air pollution problems, the Task Force envisions that most demonstrations or studies will be performed in this area. However, the data gathered and the conclusions reached will be applicable to all urban areas throughout the state where current air pollution levels exceed safe limits.

DEVELOPMENTAL STATUS AND FUTURE EFFORTS

The conclusion of the three-agency Task Force is that significant air quality benefits can be achieved with methanol replacing current uses of gasoline, diesel, and in some instances other conventional fuels. This conclusion is founded on the results of past and current demonstration projects, the results of which will be reported to the Governor in the near future.

Although the potential air quality benefits of methanol use are great, a number of barriers remain to be resolved before methanol is an integral part of California's state and regional air quality plans. The projects recommended in this report have been developed to deal with these barriers in an expedient and cost-effective manner. While past projects involving methanol have been directed at basic technology development, the focus of this report is on projects that will permit methanol technology to actually be used as an air quality strategy in areas such as the South Coast Air Basin. The recommendations in this report thus constitute the next step in bringing methanol technology from concept to commercialization. These projects will not by themselves bring about the full commercial availability of methanol as an alternative to petroleum. However, each project recommended here represents a significant step towards bringing the state's nonattainment air basins into compliance with safe and healthful clean air standards.

RECOMMENDATIONS

1. The Task Force recommends that the projects proposed in this report be approved by the Executive Officers and presented to the Boards and Commission for formal adoption.
2. The members of the Task Force believe that the cooperative task force approach to program development has been productive and successful. The Task Force recommends that this same approach be used for evaluating the proposed projects, and for developing and implementing future methanol-related activities. The Task Force should continue to meet periodically and should report to the Executive Officers annually on the results of ongoing projects and recommendations for new activities.

AIR QUALITY IMPACT OF METHANOL

Chapter 2

AIR POLLUTION IN CALIFORNIA

In spite of improvements in air quality over the past two decades, California continues to have severe air pollution problems. In the four-county South Coast Air Basin, home to nearly one-half the state's population, air pollution exceeds both national and state ambient air quality standards on 250 days or more per year. To a lesser degree San Diego, San Francisco, Sacramento, Fresno, Ventura, and Bakersfield also have unhealthy levels of air pollution.

In these urban areas, motor vehicles are by far the largest source of air pollutants. In the South Coast Air Basin alone, there are an estimated 2.3 million cars and trucks on the road every day. These motor vehicles emit hydrocarbons (unburned fuel) and oxides of nitrogen (NO_x), which chemically react in the atmosphere to form ozone, more commonly referred to as smog. Smog, which occurs on most summer days in greater Los Angeles, causes respiratory infection and eye irritation.

Motor vehicles also emit carbon monoxide, a poisonous gas which can cause angina in people with cardiovascular disease, as well as benzene, a known carcinogen. In addition diesel-powered cars and trucks emit particulates (soot), which contribute to respiratory disease, and which are a major cause of reduced visibility. Soot particles are also known to contain carcinogenic materials; whether these compounds contribute to cancer in humans is uncertain. Diesel engines also emit NO_x into the atmosphere, adding to the smog problem.

For nearly three decades, the state and counties of California have worked to reduce this air pollution. By law, new cars are equipped with emission control devices, which must be inspected for proper operation every other year. In addition, stationary sources of pollution, such as power plants and factories, are required to install equipment for controlling pollutant emissions.

These emission control programs have lowered ambient pollution levels in all of our urban areas over the last 20 years. Thanks to the effectiveness of these programs we can project attainment of clean air standards in the near future in cities such as San Francisco and Stockton.

But in other areas, such as Los Angeles, the future is not as promising. Despite reductions in the rate at which pollutants are emitted from both stationary sources and motor vehicles, the number of these sources continues to grow. Because of this growth and the severity of the current air pollution problem — which on the worst days exceeds safe levels by over 200 percent — it will be impossible to achieve clean air in greater Los Angeles (and possibly other areas of the state) unless new and innovative methods of reducing pollution are implemented. As the air quality plan for the South Coast Air Basin points out, if clean air standards are to be met, we are going to have to replace petroleum with cleaner-burning fuels whose combustion products contain less hydrocarbons, NO_x, and other dangerous pollutants. Methanol is the most likely candidate for such a clean-burning fuel.

METHANOL CAN HELP REDUCE AIR POLLUTION

With only minor changes in design, gasoline engines can burn methanol as fuel. Unlike gasoline-fueled engines, which emit many different types of hydrocarbons that react in the atmosphere to form smog, methanol-fueled engines emit only two: methanol and formaldehyde. Unburned methanol, which makes up about 90 percent of the organic exhaust emissions from methanol engines, forms smog much slower than gasoline exhaust. It is this characteristic which makes methanol fuel attractive as an air pollution control measure.

The Jet Propulsion Laboratory at the California Institute of Technology has performed an air quality modeling study to quantify the impact of replacing gasoline with methanol in California's motor vehicles. JPL found that substituting methanol-fueled vehicles for all gasoline-fueled vehicles in California will reduce the smog attributable to these vehicles by 58 percent, which would result in an overall reduction in smog of 14 percent. Another study, performed by Systems Applications, Inc. for ARCO, projected a reduction in smog of 22 percent for a similar scenario. This is a far greater reduction in smog than can be accomplished by any other feasible emission control measures currently being considered.

In addition to reducing smog, substituting methanol-fueled vehicles for gasoline-fueled vehicles would virtually eliminate emissions of benzene, a toxic air contaminant for which no safe amount in the atmosphere (threshold) is known. Approximately 90 percent of ambient benzene in the atmosphere today is attributable to motor vehicles. Methanol-fueled vehicles emit negligible benzene.

Diesel engines can also be designed to operate on methanol instead of diesel fuel, although the design changes are more extensive than those required for gasoline engines. When designed to run on methanol, a diesel engine emits 50 percent less NO_x and virtually no particulates. Technology forcing standards for diesel engines will result in development of particulate traps to control particulate emissions. These traps are not expected to reduce NO_x nor are they expected to eliminate particulate emissions. Furthermore, particulate traps that are expected to be used in the early 1990's will likely increase fuel consumption from 1 to 5 percent. Methanol-fueled diesels will not need this fuel-robbing control technology, and are expected to perform with fuel economy equal to current diesels without these control devices.

NEED FOR FURTHER STUDY

Today, enough methanol-powered vehicles have been tested for pollutant emissions to allow us to quantify emission rates with reasonable certainty. Two independent air quality modeling studies have used these emission rates to show that substantial reductions in smog can be achieved by widespread conversion to methanol fuel for light-duty vehicles. Another study, currently nearing completion at the University of California, Riverside, also indicates that smog will be reduced from methanol use in multiday as well as single-day smog episodes. Further studies of the impact of methanol on the formation of smog and other ambient pollutants are generally needed only to improve the accuracy of estimated air quality improvements.

The second pollutant emitted by methanol-fueled vehicles is formaldehyde. Formaldehyde is a very active component in the smog formation process, where it initiates ozone formation and is created as a product of photochemical reactions. Formaldehyde is also a suspected carcinogen. Methanol vehicles emit up to five times as much formaldehyde as gasoline vehicles. This raises concern that ambient concentrations of this pollutant may increase. It also suggests that if formaldehyde emissions can be reduced, additional reductions in smog are likely to be achieved. Research on improved emission control devices such as the catalytic converter to reduce formaldehyde emissions seems clearly called for, since formaldehyde is the one "red flag" associated with the environmental benefits of methanol.

THE ROLE OF METHANOL IN ACHIEVING CLEAN AIR

As indicated earlier, clean air will be achieved in the South Coast Air Basin (and several other areas of the state) only if substantial reductions in pollutant emissions are realized. The U.S. Environmental Protection Agency (EPA) requires states to be making continuous progress towards meeting clean air standards, and may withhold federal funds if ongoing progress is not demonstrated. California must seek additional ways to reduce emissions of hydrocarbons, NO_x, and other pollutants, both to reduce adverse health effects and to avoid federal economic sanctions.

Nearly all sources of pollution are currently subject to emission control requirements. More stringent emission control requirements for existing sources can be adopted, making necessary installation of more effective emission control equipment. Emission control requirements for new sources of pollution could also be strengthened, although by law the best available control equipment must currently be used. There is also some room for further reductions in emissions from new motor vehicles, and current control requirements for cars and trucks can be better enforced to achieve maximum emission reductions.

However, it is increasingly clear that even if all of these new requirements are implemented, large areas of California will still not be able to attain clean air standards. The remaining choices for further improving air quality include more costly, generally unproven emission controls; reducing industrial and population growth; and drastically reducing private vehicle usage. Implementing these types of emission control requirements is generally considered unworkable.

At this time, the most feasible alternative to such drastic measures appears to be the wider use of fuels that create less pollution than petroleum. Besides meeting this criterion, methanol has been identified as the alternate fuel which can be most easily integrated into the marketplace. The improvements in smog, ambient particulate, and benzene that would result from replacing petroleum with methanol on a large scale are significant, and would move California substantially closer to achieving clean air standards. In addition, this strategy would not carry with it the uncertainties — or the risks — associated with some of the more drastic pollution control methods listed above. When all the options are considered, methanol stands out as the most reasonable means of substantially improving the air quality in California.

The goal of the Methanol Task Force is to recommend projects which represent significant steps forward on the path to a self-sustaining, commercially viable market for methanol fuels in both the transportation and the utility/industrial sectors.

In meeting this goal, the Task Force took the approach of identifying the major barriers to methanol commercialization, and then developed strategies for removing those barriers. One obvious barrier to large-scale use of methanol is the lack of fully developed technologies (engines which burn methanol) in either the mobile or stationary sectors. Another barrier is the absence of a fueling network (the methanol equivalent of gas stations) to support methanol technology, along with a limited transportation system for bringing methanol into California. In addition, there remain a number of technical and economic uncertainties associated with the use of methanol as a fuel that need to be resolved.

The projects recommended in the next chapter are designed to address these barriers by simultaneously encouraging the development of methanol engine technology, promoting the availability of methanol as a fuel, and resolving technical and economic uncertainties.

The Task Force used the following four findings as the foundation for the recommendations included in Chapter Four:

- 1) The use of methanol in the transportation and industrial/utility sectors will result in significant air quality improvement in the South Coast Air Basin and throughout California.
- 2) The medium- and long-term supply outlook for methanol will continue to be very positive through the end of the century. This positive outlook is due to cost-competitiveness of remote and flared natural gas as methanol feedstocks and the low cost of converting coal to methanol compared with the cost of converting coal and shale oil to petroleum.
- 3) The long-term supply of petroleum at current stable prices cannot be assumed beyond the early to mid-1990's. In addition, California and United States dependence on imported petroleum will increase our vulnerability to supply interruptions in the next decade.
- 4) The broad consensus of both automotive manufacturers and energy experts is that methanol is the most viable alternative to petroleum-based fuels.

PROJECTS RECOMMENDED FOR FUNDING

Chapter 4

After a thorough evaluation of the current status of methanol technology and its potential for reducing air pollution and achieving energy independence, the Methanol Task Force recommends the following six projects:

- Project 1: Resolution of technical and economic uncertainties
- Project 2: Demonstration of flexible fueled vehicles
- Project 3: Methanol engine retrofit in transit buses (Phase I)
- Project 4: Demonstration of methanol fuel in medium-and heavy-duty trucks
- Project 5: Methanol engine retrofit in transit buses (Phase II)
- Project 6: Economic assessment of methanol use in cogeneration turbines and utility boilers

The first three projects are recommended for immediate funding and are summarized in Table 1. Detailed descriptions of Projects 1 through 3 are presented at the end of this chapter.

The second group of projects are recommended should funding become available. These three projects are summarized in Table 2 with detailed descriptions also following.

Each of these projects has been specifically developed as a response to the barriers which currently exist to the commercialization of methanol as a fuel. While these six projects will not bring about the full commercial availability of methanol, each represents a major step towards substituting methanol for conventional petroleum-based fuels.

Table 1. Projects Recommended for Funding

Project	Objectives	Cost (\$K)	Responsible Agency	Source of Funding (\$K)	Start Date (FY)	Duration (yr)
1. Resolution of technical and economic uncertainties						
a. Summary of air quality benefits	<ul style="list-style-type: none"> Define and determine benefits for potential methanol use scenarios Determine magnitude and source of uncertainties; produce workplan to address removing uncertainties 	150	ARB ^a	FY85/86 Research Budget	1986	1
b. Development of low-formaldehyde methanol passenger car controls	<ul style="list-style-type: none"> Determine lowest achievable formaldehyde emissions from LDVs (current and advanced technologies) 	150	ARB ^a Catalyst and auto industries	FY86/87 Research Budget —	1987	1
c. Study of economic incentives and funding sources to support expanded use of methanol	<ul style="list-style-type: none"> Determine potential for emission offsets and other economic incentives to encourage methanol use Determine potential sources for funding continued demonstration of methanol technologies 	75	SCAQMD ^a CEC	Unobligated Reserve (45) ERF ^b (30)	1986	1
2. Evaluation of flexible fueled vehicles (cars and light-duty trucks)	<ul style="list-style-type: none"> Demonstrate technology Overcome fuel network limitations Assess emissions impact Expand model availability 	216	ARB ^a CEC SCAQMD	Equipment Budget (72) ERF ^b (72) Unobligated Reserves (72)	1986	2
3. Methanol engine retrofit in transit buses (Phase I)	<ul style="list-style-type: none"> Demonstrate feasibility of retrofit (new engine) Determine operational requirements Provide experience necessary to develop a low cost retrofit kit 	975	ARB ^a GM RTAC ^c Celanese	Existing Resources Settlement (975) In kind Fuel	1986	4

^aLead agency

^bEnergy Resources Fund

^cRiverside Transit Agency

Table 2. Projects Recommended If Additional Funding Becomes Available

Project	Objectives	Cost (\$K)	Responsible Agency	Source of Funding (\$K)	Start Date (FY)	Duration (yr)
4. Demonstration of methanol fuel in medium- and heavy-duty trucks	<ul style="list-style-type: none"> • Develop technology • Reduce diesel pollution • Increase types of vehicles able to use methanol 	2200	CEC ^a ARB	TBD ^b	TBD	5
5. Methanol engine retrofit in transit buses (Phase II)	<ul style="list-style-type: none"> • Develop retrofit kit; demonstrate on larger number of buses • Reduce diesel pollution by accelerating substitution of methanol for diesels • Expand transit district fuel and emission control options 	3000	ARB ^a CEC	TBD	1989 if Project 3 (Table 2) is successful	5
6. Economic assessment of methanol use in cogeneration turbines and utility boilers	<ul style="list-style-type: none"> • Assess emission benefits • Evaluate life-cycle cost for various applications • Determine feasibility end-uses and develop implementation plan 	250	SCAQMD ^a CEC	TBD	TBD	TBD

^aLead agency

^bTo be determined

In general, technology evolves through six stages to achieve a sustainable, long-term market as indicated in Figure 1.

In making its recommendations, the Methanol Task Force has taken into account the current stage of development of the various types of methanol technologies. Thus, while limited commercial demonstrations (Stage 4) of methanol cars have been underway in California for the last several years, much less work has been done on other types of vehicles, such as buses and trucks. Likewise, in the stationary sector, very little methanol research and development has been undertaken. Table 3 presents the current stage of development of methanol technology for different types of vehicles and indicates how each type of vehicle would be affected if the Task Force recommendations are implemented.

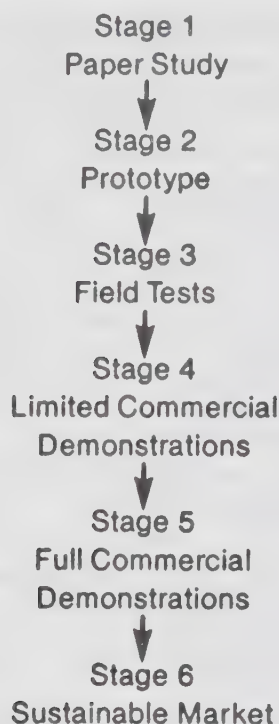


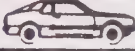

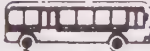




Figure 1. Six Stages of Technology Evolution.

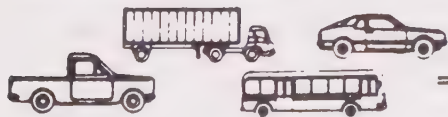
The Task Force also recognized, that to be effective, any program aimed at developing an alternative to petroleum must be able to displace the majority of refined products from crude oil. Since gasoline comprises only 45 percent of the refined products from each barrel of oil, a program to replace petroleum fuels must also address diesel and fuel oil use as well.

The projects proposed by the Methanol Task Force are designed to continue the public/private partnership approach to funding these types of projects. This cost-sharing approach leverages state money and increases the commitment of the participants. The projects recommended here also protect and build on the prior investment in methanol technology by the state and private industry. Each project is intended to utilize the results of previous methanol demonstration projects and to develop the technology and the market for methanol in a logical and consistent manner.

Table 3. Status of Methanol Vehicle Technology

Vehicle Type	Paper Study	Prototype	Field Tests	Limited Commercial Demonstration	Full Commercial Demonstration	Sustainable Market
	Example: Concept Plan	Example: Chassis dynamometer test, track test and limited field testing, 1 to 2 vehicles	Example: Controlled fleet test, modified production run, 5 to 30 vehicles	Example: Multiple fleet demonstration, 500 to 2,000 cars produced on the assembly line	Example: Full-scale introduction into specific markets, e.g., government fleets; 5,000 to 10,000 vehicles	Example: Multiple market segment including private vehicles, full-scale service network, resale market, model choice, 20,000 to 50,000 vehicles/year
Autos and Light Trucks		FFVs 	Phase I Project 6 FFVs			
				Methanol Cars 		
	Low formaldehyde Methanol Car 	Phase I Project Low formaldehyde research vehicle				
Buses		New Buses 	SCRTD Project* 30 Buses			
	Existing Buses 	Phase I Project 3 RTA Buses	Phase II Project 30 Re-engined Buses			
Trucks	Interstate 	Phase II Project 5 Heavy Duty Trucks				
	Urban Trucks 	Phase II Project 5 Medium Duty Trucks				

*Project funded solely by SCRTD



= Current status of technology

→ = How status will change due to proposed project

PROJECT 1 — RESOLUTION OF TECHNICAL AND ECONOMIC UNCERTAINTIES

Project Objectives and Rationale

Research completed by ARB and CEC over the past 5 years has shown that substituting methanol for petroleum fuels in California vehicles is one of the most promising strategies for solving the state's continuing air pollution problems. However, certain technical and economic questions still need to be answered to give government and industry the confidence to make a long-term transition from petroleum to methanol. In addition, more information is needed about methanol as a fuel in both gasoline and diesel engines to guide vehicle manufacturers and fuel producers in research and development.

To resolve these remaining uncertainties about the viability of methanol, research is needed to establish:

1. A more exact quantitative estimate of the air quality benefits of methanol fuel use in both diesel and gasoline engines;
2. The optimal approach to minimizing cold-start formaldehyde emissions from methanol vehicles, as well as guidelines for future formaldehyde and unburned methanol emission standards;
3. The most effective economic incentives and funding sources to support precommercial research, development, and demonstration.

Proposal

The study to quantify air quality benefits of methanol fuel use will include:

- The air quality benefits for specific potential methanol use scenarios;
- The magnitude and source of uncertainties in these estimates;
- A work plan to eliminate these uncertainties;
- A nontechnical summary report on the benefits of methanol fuel use.

The study on minimizing formaldehyde emissions from methanol use will investigate potential strategies for achieving maximum reasonable reduction of formaldehyde emissions from light-duty vehicles (LDV). The EPA, in cooperation with the engine and emission control industries, is currently investigating the control of formaldehyde emissions in heavy-duty vehicles. The Task Force expects that EPA and industry will participate in an evaluation of LDV formaldehyde emissions control options with the state.

The final study of this proposal will investigate economic incentives and funding sources for sustained methanol development. This study will explore the potential for emission offsets and other regulatory and fiscal incentives to encourage the use of methanol, as well as sources of funding and public/private cost-sharing to bring methanol technologies to the commercialization stage.

Funding

- Air quality benefits — \$150,000, ARB (85/86 FY research budget)
- Formaldehyde control options — \$150,000, ARB (86/87 FY research budget); automotive industry; emission control industry
- Incentives and funding sources — \$75,000, CEC (ERF, \$30,000); SCAQMD (unobligated reserves, \$45,000)

Schedule

- Air quality benefits — March 1986, RFP issued; March 1987, study completed
- Formaldehyde control options — August 1986, RFP issued; August 1987, study completed
- Incentives and funding sources — August 1986, RFP issued; August 1987, study completed

Responsible Agency

- Air quality benefits: ARB
- Formaldehyde control options: ARB
- Incentives and funding sources: SCAQMD and CEC

PROJECT 2 — EVALUATION OF FLEXIBLE FUELED VEHICLES (FFVs)

Project Objectives and Rationale

As shown in Table 3 (Status of Methanol Vehicle Technology), the technology for using methanol as a fuel in light-duty vehicles has already reached a mature level. Development of prototypes, field tests, and limited commercial demonstrations have all been successfully carried out. However, large-scale commercial demonstrations are not currently feasible because methanol is not widely available at commercial refueling stations. One promising solution to this transitional "chicken and egg" problem is the recent development of prototype flexible fueled vehicles (FFVs) which can operate on any combination of fuel methanol or gasoline. FFVs can be designed in a wide range of body types, from small economy cars to large luxury models, and are built to give optimum performance when fueled with methanol. Even though they are designed to burn methanol, FFVs can also be fueled with gasoline if methanol is not available.

The obvious advantage of vehicles capable of burning either gasoline or methanol is that they could be marketed in California without the need for a fully developed methanol fuel network. Once a substantial pool of methanol-compatible vehicles is introduced, substituting methanol for gasoline or diesel fuel will become more economically viable.

While FFV technology has the potential for overcoming several significant barriers to the commercial use of methanol as a vehicle fuel, the technology is still in the developmental stages, and additional demonstration and testing are needed.

One major advantage of methanol-fueled vehicles is that their exhaust hydrocarbons are much less photochemically reactive than gasoline vehicle exhaust. While evaporative and exhaust emissions have been studied from vehicles fueled with pure or nearly pure methanol and gasoline, little or no testing has been done on vehicles fueled with blends closer to the middle of the range (40 to 80 percent methanol).

The research proposed here will make it possible to assess the emission durability, exhaust reactivity, and evaporative emission levels of a wide variety of gasoline-methanol blends in FFVs.

The California Energy Commission has successfully demonstrated that field testing a limited number of methanol-fueled vehicles as part of various state and local government fleets can supply valuable performance and emissions data. The early fleet of methanol Escort station wagons operated by Los Angeles County continues to provide, for instance, valuable high mileage information on the material compatibility constraints for methanol vehicles. Similar relevant details expect to be generated by a fleet of six FFVs operated by the ARB, CEC, and SCAQMD.

Proposal

The Task Force proposes testing a small fleet of flexible fueled vehicles. Each of the participating agencies would purchase and operate two FFVs. Performance, durability, and emissions would be evaluated. The FFVs would be emission tested by the ARB, and technical support would be provided by the CEC.

Funding

- ARB — \$72,000
- CEC — \$72,000
- SCAQMD — \$72,000

Schedule

- December 1986 — FFVs delivered
- December 1988 — evaluation completed

Responsible Agency

- ARB, with assistance of CEC and SCAQMD

PROJECT 3 — METHANOL ENGINE RETROFIT IN TRANSIT BUSES (PHASE I)

Project Objectives and Rationale

The contribution to air pollution from transit buses is a small part of total heavy-duty mobile source emissions. However, the fact that buses run in densely populated urban areas subjects large numbers of people to localized high concentrations of unhealthy diesel exhaust emissions. If transit buses can be converted to operate on methanol, particulate and nitrogen oxide emissions will be substantially reduced from the levels currently produced by diesel bus engines. Thus, methanol technology has the potential for solving another pollution problem in our large cities.

Transit buses have a lifespan of more than 15 years. Because of reductions in federal funding for purchases of new buses, the useful life of existing transit buses is expected to be extended through engine rebuilding or replacement and chassis refabrication. This will decrease the air quality improvement that could be achieved by replacing older diesel engines with new methanol engines. On the other hand, the longer lifespan of the buses offers an opportunity for converting existing buses to cleaner burning methanol during a major rebuild. This project will provide the initial prototype assessment of this concept and establish an engineering baseline for development of a retrofit kit.

Proposal

Three recent model buses currently in operation at the Riverside Transit Agency (RTA) will be modified to burn methanol. The buses will be placed in revenue service to demonstrate the feasibility of this concept. Modifications to the buses will include installation of methanol-compatible fuel tanks and fuel systems, revisions to the electrical system, and installation of the latest version of a Detroit Diesel Allison-General Motors methanol bus engine. One bus will receive extensive track testing prior to being placed into revenue service. Bus exhaust emissions will be measured.

During revenue service, the performance, fuel economy, maintenance and repair requirements, and public acceptance of the buses will be evaluated. The study will provide information which can be used to further refine the methanol engine, and will establish the operational requirements for methanol buses. This experience will also form the basis for future development of a lower cost retrofit kit for converting buses to methanol during rebuilding. (A follow-on project to evaluate this kit on a larger number of buses is also proposed. See Project 5).

Funding

This project has been initiated. General Motors will provide the retrofit to methanol of three transit buses, as well as field support for operation of the buses for three years. The cost of this part of the project will be paid by General Motors as settlement for an unrelated enforcement action. The Riverside Transit Agency (RTA) will provide the three transit buses and operate them for three years. The Celanese Corp. has agreed to provide the methanol. The transit agency already has a fueling system which is compatible with methanol. The ARB will coordinate the effort.

- Retrofit of buses — \$975,000, General Motors
- Operate buses — in kind, Riverside Transit
- Provide fuel — Celanese Corp.
- Oversee project — ARB

Schedule

- Project initiation — December 1985
- Begin retrofit of first bus — July 1986
- First bus enters service — March 1987
- Initial report — May 1988
- Project complete — August 1990

Responsible Agency

- ARB

PROJECT 4 — DEMONSTRATION OF METHANOL FUEL IN MEDIUM-AND HEAVY-DUTY TRUCKS

Project Objectives and Rationale

Diesel engines are a major source of particulate and oxides of nitrogen (NO_x) emissions. They are thus a major contributor to levels of ozone, nitrogen dioxide, and fine particulates that exceed California's ambient air quality standards. In addition, particulates from diesel engines reduce visibility, and are a major source of public complaint. Using methanol in diesel engines has the potential to virtually eliminate particulate emissions and reduce NO_x emissions by one-half. This makes methanol a more effective pollution control strategy than any diesel emission control device currently available or being developed.

The use of methanol as a fuel has been successfully demonstrated in two large diesel bus engines currently in revenue service in Marin County north of San Francisco. Methanol has not been demonstrated to date, however, in any of the other types of diesel engines commonly used in heavy-duty trucks.

Because heavy-duty trucks are responsible for the majority of diesel-related pollution, it is important to advance the development of methanol engine technology for these vehicles. The objective of this project is to advance methanol heavy-duty truck engine technology to the prototype stage by evaluating the use of methanol in both two-stroke and four-stroke diesel truck engines.

Proposal

Five of each of these engine types will be placed in service in trucks operating in the Los Angeles area. The fuel economy, operability, and serviceability of each engine will be investigated over a 3-year period. The project will provide the information necessary for fleet operators to consider more extensive demonstration of methanol trucks, and will expand the types of methanol-diesel engines available for trucking applications.

The cost of the project will be shared by the state, engine manufacturers, and the trucking industry. The state will purchase five medium-duty and five heavy-duty trucks (delivery vans, garbage trucks, or petroleum tank trucks) with engines designed to operate on methanol. These trucks will be placed in operation in commercial or government fleets. The state will also provide a fuel and maintenance subsidy for the first three years of operation. Each of the fleet operators will be responsible for providing fuel and maintenance, and for keeping records of vehicles' operation. Methanol engines will be developed by interested engine manufacturers.

Funding

Project cost will be \$2.2 million in state funds, with the engine development and operational costs provided by the engine manufacturer and fleet operators, respectively. A funding source for this project has not been identified.

Capital Equipment	\$1,400,000
Fuel/Maintenance	450,000
Technical Support	350,000
Total	\$2,200,000

Schedule

Initiation (I)	Present detailed proposal to Commission for approval
I + 2 years	Trucks enter fleet service
I + 3 years	First annual report
I + 5 years	Demonstration complete

Responsible Agency

CEC, with assistance of ARB

PROJECT 5 — METHANOL ENGINE RETROFIT IN TRANSIT BUSES (PHASE II)

Project Objectives and Rationale

This project will take advantage of the findings and experience gained in Project 3 to advance methanol bus retrofit technology from the prototype to field test stage of development (see Table 3). By expanding the number of coaches retrofitted to methanol, a statistically valid determination of durability and operational expense will be gained. This is a prerequisite to a full commercial demonstration of this technology. In addition, the cost of the retrofit will be lowered by developing an optimal methanol conversion kit based on the experience of Project 3.

Proposal

Thirty transit buses will be retrofitted to burn methanol fuel. These retrofitted coaches will be placed in revenue service in two transit districts selected on the basis of fleet size, availability of routes in high density urban areas, maintenance practices, and availability of appropriate coaches. The buses will be retrofitted for methanol fuel at the time of a scheduled engine rebuild. Since the Detroit Diesel Allison (DDA) 6V-92TAC engine is used in one-third of the buses presently operating in the South Coast Air Basin, the project will utilize the methanol version of this engine. Retrofit kits for two different transit coach models will be engineered, and some of each model will be retrofitted. The coaches will be retrofitted using either rebuilt, remanufactured, or new methanol engines, depending on the type of kit developed. The buses will be operated over a 2-year period so that the reliability and durability of the engines can be established.

Funding

New methanol engines will cost about \$25,000 each; rebuilt or remanufactured engines somewhat less. Modifications to the buses, including catalytic converters, will add approximately an equal amount to this cost. A three year program, including installation of two methanol fueling facilities and differential operating and maintenance costs, will cost approximately \$3 million. A funding source for this project has not been identified.

Schedule

1989, if a funding source is identified.

Responsible Agency

ARB, with assistance of CEC

PROJECT 6 — ECONOMIC ASSESSMENT OF METHANOL USE IN COGENERATION TURBINES AND UTILITY BOILERS

Project Objectives and Rationale

Tests conducted by Southern California Edison and the California Energy Commission have shown that burning methanol in utility boilers and in utility/industrial gas turbines (including cogeneration facilities) can significantly reduce emissions of NO_x and SO_x compared to conventional fuels. The SCAQMD has recently established the most restrictive Best Available Control Technology (BACT) requirement on cogeneration projects. Methanol use in cogeneration and utility facilities may offer an important option for meeting BACT emission limits while avoiding the large capital investment of Selective Catalytic Reduction (SCR) control technology.

The major barriers to the use of methanol in stationary sources are the higher price of methanol compared to natural gas, coupled with the lack of commercial experience with methanol. In addition, methanol suppliers are reluctant to guarantee prices for multiyear periods. Before proceeding with further stationary methanol demonstration projects, it is necessary to explore future scenarios that might close the price gap between methanol and natural gas.

The current delivered price of methanol is inflated by the high cost of transporting small volumes of methanol in rail tank cars, thus limiting the potential of methanol as an industrial fuel. Strategies must be found for transporting methanol in large bulk shipments, so that a more competitive and stable price of methanol can be established. This could in turn attract significant levels of investment on the part of vehicle and industrial equipment manufacturers, fuel producers, and marketers.

Major uncertainty exists among turbine manufacturers, fuel users, producers, and governmental agencies regarding the life-cycle costs of methanol as a cogeneration or utility fuel. This uncertainty has kept commercial operators from assessing the performance and cost of methanol under operating conditions. An economic assessment is thus needed to compare the life-cycle cost of methanol-fueled cogeneration and peaking gas turbine facilities to similar facilities fired by natural gas and equipped with SCR. Such an assessment will make it possible to accurately determine whether or not there is a point at which methanol is an economically viable option. The long-term cost effectiveness of methanol use as an emission reduction strategy also needs to be compared against natural gas with SCR.

Also needed is an economic assessment of an offset fee which could be paid by cogenerators to a utility through a Public Utility Commission (PUC) transmission line fee assessment so that a utility could cost effectively overfire methanol in its boilers. This fee

would provide for net emission reductions of NO_x based on the fuel cost differential for new cogeneration facilities unable to meet strict NO_x limits. To undertake an analysis, detailed information on the capital and operating costs and technical feasibility of overfiring in utility boilers will be needed. State funding through the Energy Technology Research, Development, and Demonstration Program (Rosenthal, Naylor Act of 1984) should be considered as a potential cofunding option for this test. SCE is proposing to undertake utility-scale tests of this type in late 1986; this analysis should be coordinated with these tests.

The increased market demand for methanol in the stationary source sector could lower methanol costs by lowering incremental shipment and storage costs. However, the size of the methanol purchase required and the possible cost reduction has not been analyzed. An assessment is needed to determine the conditions under which methanol costs can be reduced. This assessment would consider the commercial viability of methanol in terms of aggregate demand, shipment logistics, storage costs, and infrastructure development.

Proposal

A study will be performed of the major economic issues affecting the use of methanol in utilities, cogeneration turbines, and other stationary sources, including:

- The long-term costs and control effectiveness of methanol as compared to natural gas with Selective Catalytic Reduction;
- The feasibility of a PUC-based fuel cost differential fee to be paid to utilities to use methanol in exchange for emission offsets;
- The feasibility of methanol overfiring at a utility boiler;
- The impact of large-scale demand for methanol on the long-term cost of methanol.

Funding

\$250,000 — To be determined between CEC and SCAQMD

Schedule

To be determined

Responsible Agency

SCAQMD, with assistance of CEC

SAE Technical Paper Series

872093

Comparative Study of Heavy-Duty Engine Operation with Diesel Fuel and Ignition-Improved Methanol

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International Fuels and Lubricants
Meeting and Exposition
Toronto, Ontario
November 2-5, 1987

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Comparative Study of Heavy-Duty Engine Operation with Diesel Fuel and Ignition-Improved Methanol

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ABSTRACT

Methanol can be made suitable for compression ignition engines by ignition-improving additives. The ignition improver demand can be minimized by increasing the compression ratio.

The technical suitability of this fuel can be regarded as proven, since most of the problems connected with its use have been solved. Its economic viability, however, has still to be doubted.

From an environmental point of view, ignition-improved methanol deserves great interest due to the total absence of soot in the exhaust and the considerably reduced NO_x emission.

HEAVY-DUTY VEHICLES are almost exclusively fitted with diesel engines. It would be highly desirable to eliminate their principal drawback, i.e. the emission of particulate matter, without sacrificing their benefits in respect of fuel efficiency, useful life, serviceability etc.. Methanol would be an ideal substitute for diesel fuel because of its extremely low soot formation tendency (1)*, if its inadequate ignition quality did not prevent its straight use in compression ignition engines.

Due to its high octane number, methanol is best suited for spark ignition engines. In this case, it is even possible to achieve an certain recovery of lost heat (2,3). Nevertheless, a considerable

fuel consumption penalty has to be accepted, when compared to the use of methanol in compression ignition engines (4).

Therefore, it appears more sensible to combine the technically advantageous features of the diesel engine with the environmentally attractive ones of methanol. This can be achieved by the addition of so-called ignition improvers (5). It is noteworthy that ethanol with thus enhanced ignition quality is already in commercial use in Brazil (6,7).

OPTIMIZATION OF IGNITION IMPROVER CONTENT

For use in alcohols, nitrates are the most effective ignition improvers (8,9), and triethylene glycol dinitrate (Tri-EGDN) is one of the most advantageous (Fig. 1). Nevertheless, 2-ethylhexyl nitrate (under the trade name DI13 a product of Ethyl Corp., USA) was used in part of the tests. A third ignition improver (a proprietary product of AECL, Ltd., South Africa) was applied in endurance tests (10,11).

When using alcohols in diesel engines, an ignition improver is required, along with a whole range of other additives:

- So far, 1% by volume (1.2% by mass) castor oil has been used to avoid excessive wear of injection system components. But promising results have been obtained from work carried out to develop more cost-effective lubricity additives to be used in much lower concentrations.

- 0.02% by volume morpholine, giving a pH of at least 7 (12,13), was used as a corrosion inhibitor (10). This is required to protect the injection system from the corrosive effect of methanol, which is intensified by the ignition improver.

Further unfavorable characteristics of methanol are its low boiling temper-

* Numbers in parentheses designate references at end of paper

Fig. 3: Ignition improver content and rate of pressure rise

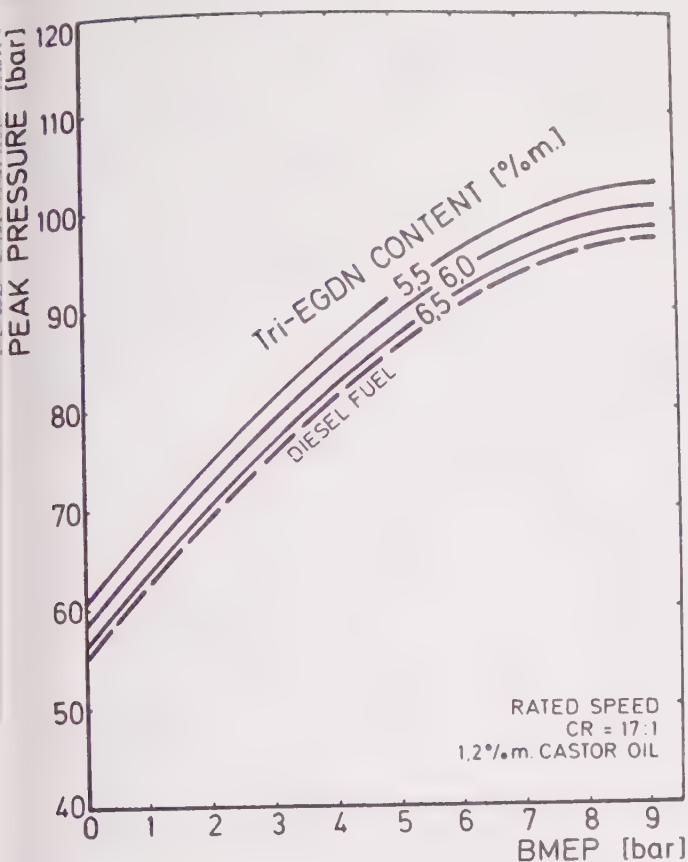


Fig. 4: Ignition improver content and peak cylinder pressure

engine wear and greater combustion noise. Concentrations lower than those stated in Fig. 2 resulted in engine misfiring.

With an ignition improver concentration of 6% by mass, the methanol-fuelled engine started from cold with slightly more difficulty than the diesel-fuelled one. During the initial warm-up, the engine ran roughly for only about one minute. Afterwards, idling was normal and restarts were instantaneous without any hot-start problems.

The lower heat value of methanol fuel is less than half that of diesel fuel (Fig. 5). In order to introduce the same amount of energy into the combustion chamber, 2.23 times to volume must be injected. This requires enlarged flow cross sections in the injection nozzles, which usually can be achieved without difficulty. Unfortunately, however, available injection pumps cannot always be adapted to the larger injection volume. In the majority of cases, naturally-aspirated engines can be converted to methanol operation without significant loss of power. But this usually is impossible to achieve with turbocharged engines, since the quantity of diesel fuel delivered is already much higher in this case.

Therefore, it will not always be

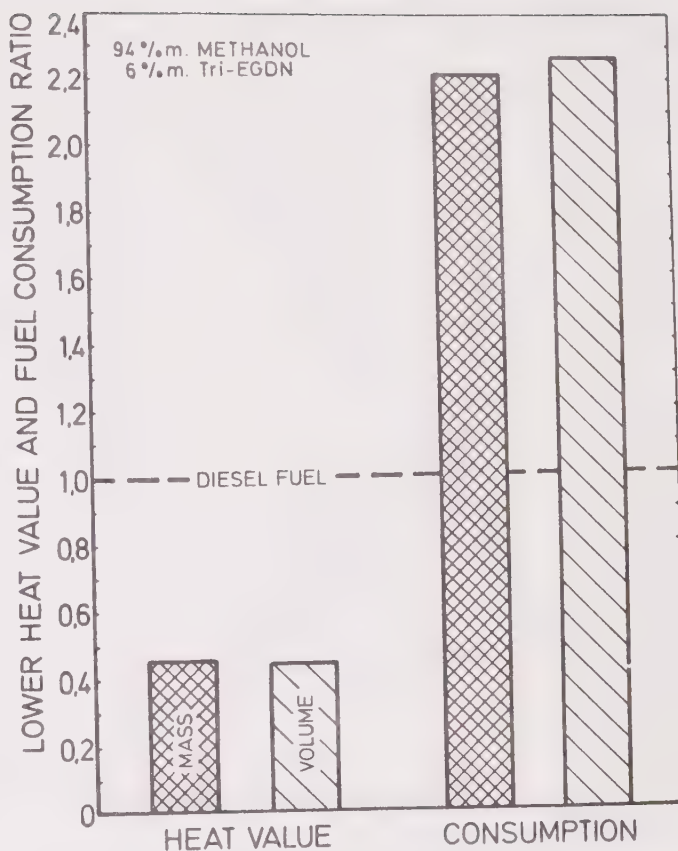


Fig. 5: Lower heat value and consumption of ignition-improved methanol

possible to convert engines with off-the-shelf components. Introduction of ignition-improved methanol for heavy-duty diesel engines would necessitate the design of new injection pumps in most cases.

THERMODYNAMIC CONSIDERATIONS

Fig. 6 presents the results of comparative calculations of the ideal diesel engine cycle. The thermal efficiency with methanol and diesel fuel is virtually identical if the air/fuel ratio is the same. The mean effective pressures, however, are 4 to 5% higher for methanol, as its ratio of lower heat value to stoichiometric A/F ratio is higher by the same percentage. Thus, with the same MEP, the efficiency of methanol combustion is correspondingly greater. Unfortunately, however, this advantage is smaller, the lower the MEP, i.e. the larger the air/fuel ratio.

Accordingly, it follows from Fig. 7 that the indicated and brake thermal efficiencies with both fuels differ only at high engine load. With methanol fuel, however, significantly higher brake mean effective pressures can be achieved. This is due to the fact that methanol combustion does not produce any black smoke,

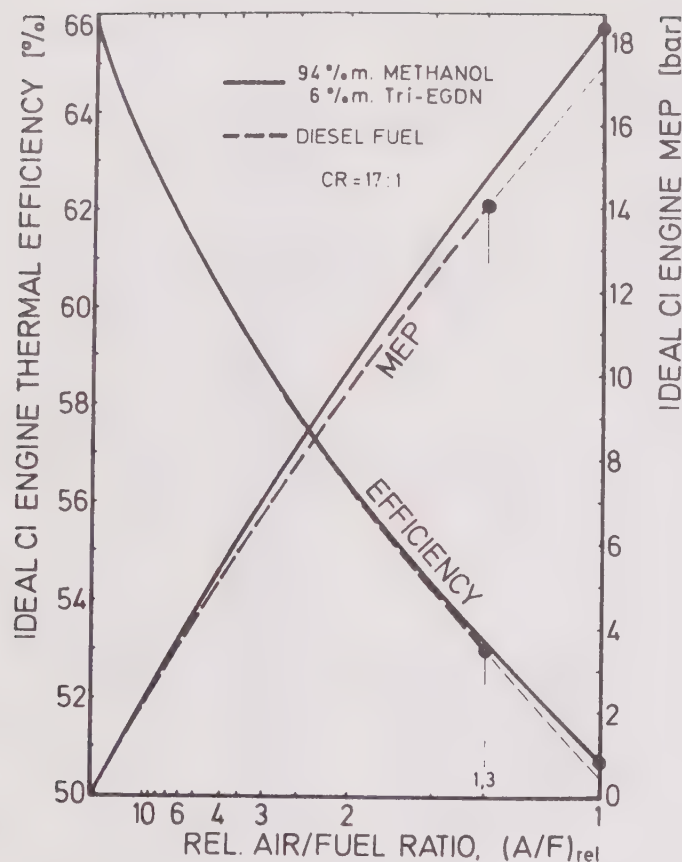


Fig. 6: Ideal diesel engine efficiency and MEP with methanol and diesel fuel

whereas soot formation properties of diesel fuel limit the relative A/F ratio to approximately 1.3, i.e. 30% air excess over stoichiometric.

However, possibility of operating the methanol-fuelled engine at reduced A/F ratios was not pursued. Therefore, the present comparison of diesel-fuelled and methanol-fuelled engines is based on injection pump settings for the same rated power which implies practically the same A/F ratio. The power output with ignition-improved methanol was slightly higher at low engine speeds (Fig. 8), as a result of the fuel delivery characteristics of the injection pump.

The full load test results in Fig. 8 show that the actual efficiency advantage of methanol, approx. 5%, was greater than could be expected from the aforementioned calculations (approx. 2.5%). This may be due to higher rates of heat release during methanol combustion which could not be taken into account.

MINIMIZATION OF IGNITION IMPROVER CONTENT

The ignition improver concentration required, 3.5% by volume (6% by mass) of Tri-EGDN, is relatively high with compression ratios optimum for diesel fuel

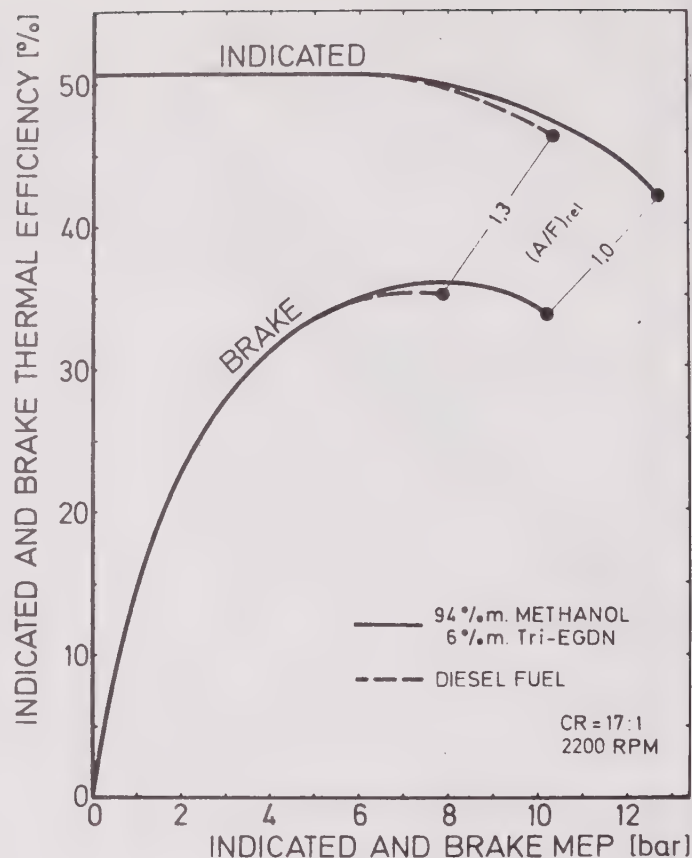


Fig. 7: Indicated and brake MEP and corresponding efficiencies with methanol and diesel fuel

use, e.g. 17:1. Therefore, ignition improver content is the decisive factor in determining fuel costs and thereby the economic viability of ignition-improved methanol.

The ignition improver demand can be significantly reduced by increasing the compression ratio, as is shown in Fig. 9 for Tri-EGDN and 2-ethylhexyl nitrate. The percentage was matched to a level aimed at retaining smooth engine running with no misfiring and no cyclic variations in cylinder pressure. In all cases, the start of injection was adjusted for minimum fuel consumption.

The straight lines in Fig. 9 result from linear regression analysis. The intersection at 25.3:1 represents the lowest compression ratio which permits engine operation without ignition improver. Such a high compression ratio is not suitable for practical application, as the resultant high peak cylinder pressures would increase mechanical engine loads, friction losses, fuel consumption and wear to an impermissible extent.

With diesel fuel, the ignition delay decreases considerably as the compression ratio increases (Fig. 10). For minimum fuel consumption, the start of combustion

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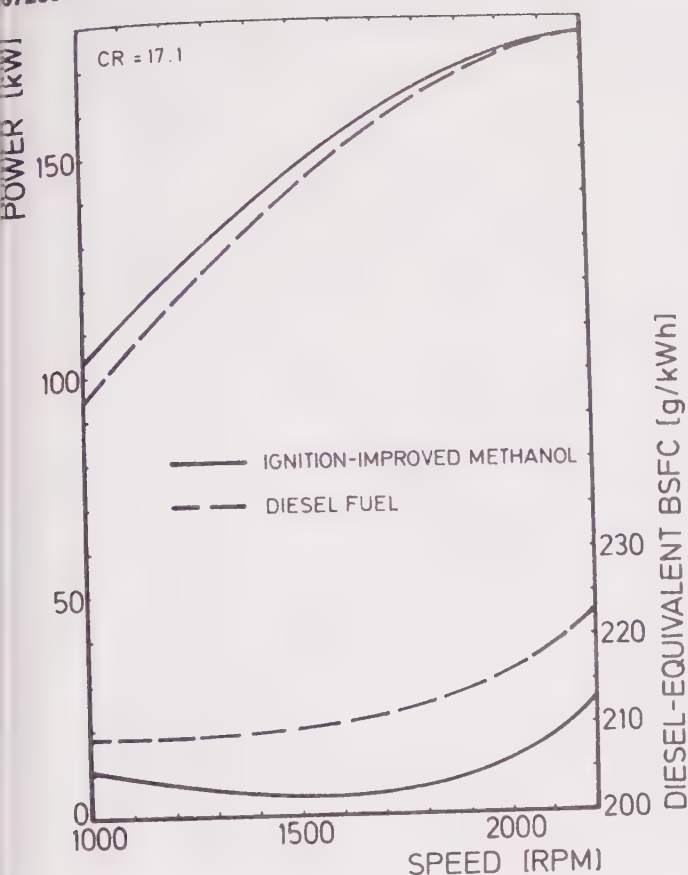


Fig. 8: Full load performance with diesel and methanol fuel

has to be advanced, which implies a correspondingly retarded start of injection. Methanol fuel necessitates a different start of combustion which incidentally results in the same start of injection for both fuels. The same ignition delay is only achieved at a compression ratio of approximately 15:1.

Higher compression ratios increase the peak cylinder pressures quite considerably. The relatively high pressures in Fig. 11 (as those in Figs. 3 and 4) result from an injection timing for minimum fuel consumption. Considerably lower peak pressures could be attained by retarding the injection timing and accepting slightly higher fuel consumption.

ENVIRONMENTAL ASPECTS

The formation of nitrogen oxides with methanol fuel is determined by superposing effects. It is promoted by increasing the compression ratio, and countered by retarding the injection timing (13). In fact, Fig. 12 reveals a NO_x reduction which implies an overcompensation of the temperature-increasing effect of the compression ratio by the temperature-reducing effect of the retarded injection.

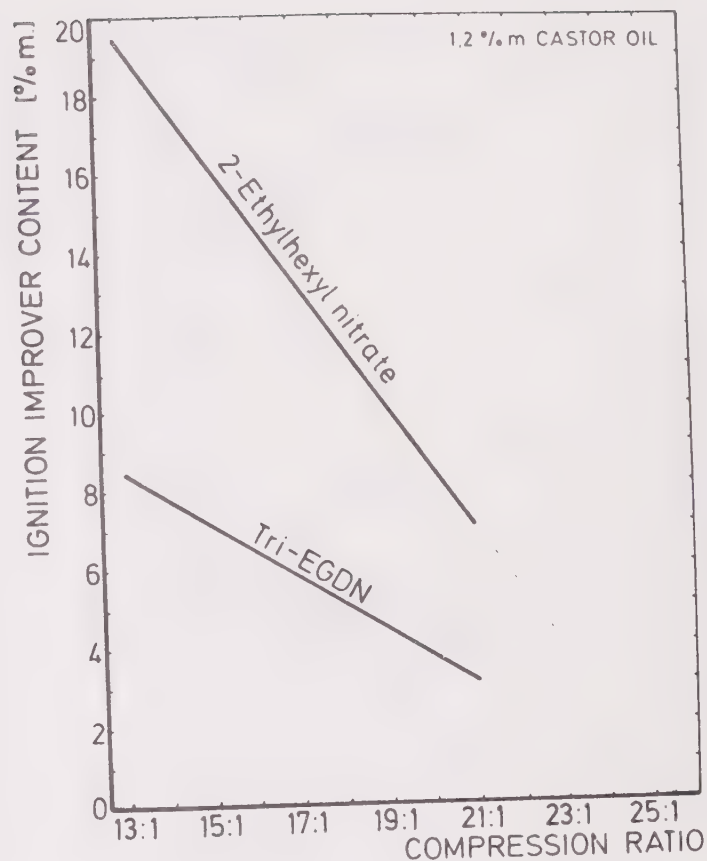


Fig. 9: Compression ratio and ignition improver required

The NO_x concentration in the exhaust is considerably lower with methanol than with diesel fuel, especially as engine load rises. Reasons for this are the retarded start of combustion and the greater specific heats of the combustion gases. With the same heat release, both result in lower combustion temperatures and thereby reduced NO_x formation.

The lower content of ignition improver is not likely to have significantly contributed to the NO_x emission reduction, since the nitrogen portion of the nitric acid ester used is largely released in elemental form. For the same reason, the use of nitrates does not cause an excessive increase in NO_x emission, as is sometimes anticipated.

Fig. 13 shows the corresponding carbon monoxide emissions. With decreasing load, they are increased because of the drop in combustion temperatures and the resultant shift in the water/gas equilibrium. For obvious reasons, this tendency is intensified with methanol fuel and decreasing compression ratio. At high engine loads, CO concentration increases sharply, primarily due to the reduced air/fuel ratio. The fact that concentrations are less with methanol fuel is attributable to its more favorable ratio of

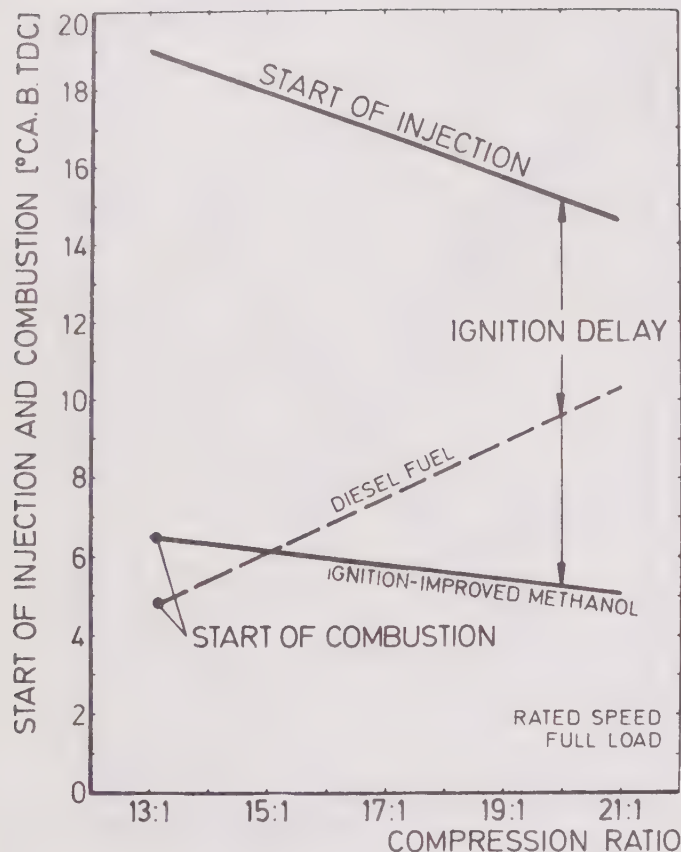


Fig. 10: Injection timing and ignition delay with diesel fuel and ignition-improved methanol versus compression ratio

calorific value to stoichiometric mass of air per unit mass of fuel (10). The significant increase in CO concentration with rising compression ratio results from the geometrical reduction of the piston bowl volume and thus of the mass of air present therein and available for combustion.

The concentrations of unburnt hydrocarbons (Fig. 14) rise significantly with falling BMEP and lower compression ratio. Therefore, increased compression is highly recommended when burning ignition-improved methanol.

The primary advantage of ignition-improved methanol, however, is the total absence of soot in the exhaust gas. For this reason, and also because of low nitrogen oxide emission, this fuel represents an interesting alternative for diesel engines, when considered from an environmental point of view. The additional application of high compression ratios would not only lower fuel costs by reducing the ignition improver requirement, but would also lower NO_x and HC emissions without having to accept any impermissible increase in the CO emission.

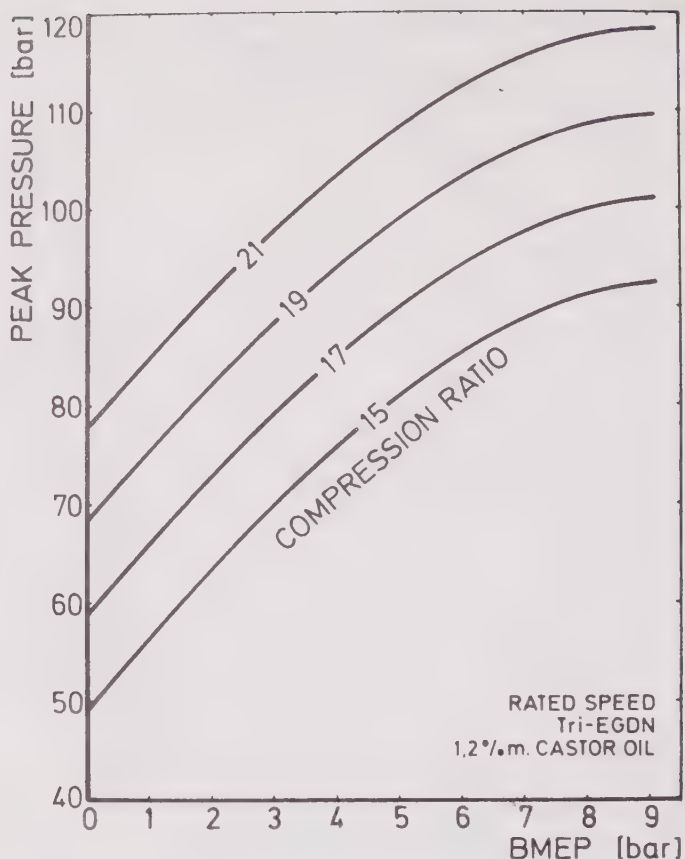


Fig. 11: Compression ratio and peak cylinder pressure

EMISSION TEST RESULTS

For gaseous exhaust emission testing, the 13-mode cycle was applied, as the single cylinder laboratory engine used for the present investigation did not permit transient tests to be carried out. This does not represent a serious disadvantage, however, as the emission which can be measured only in the transient test, namely particulate matter, is extremely small due to the soot-free exhaust gas typical of methanol combustion. Also, measured values obtained from transient tests are questionable because of the great measurement error at very low PM emission levels. Based on results from another test program, it can be concluded that the particulate matter emission would have been about 0.07 to 0.09 g/BHP-h, compared to 0.55 g/BHP-h with diesel fuel. This represents a reduction in the PM emission of more than 80%.

The superiority of ignition-improved methanol over diesel fuel with regard to gaseous exhaust emissions is illustrated in Fig. 15. The test results were obtained from 13-mode cycles carried out with methanol fuel and 17:1 as well as 21:1 compression ratios and are compared to the emission levels achieved with die-

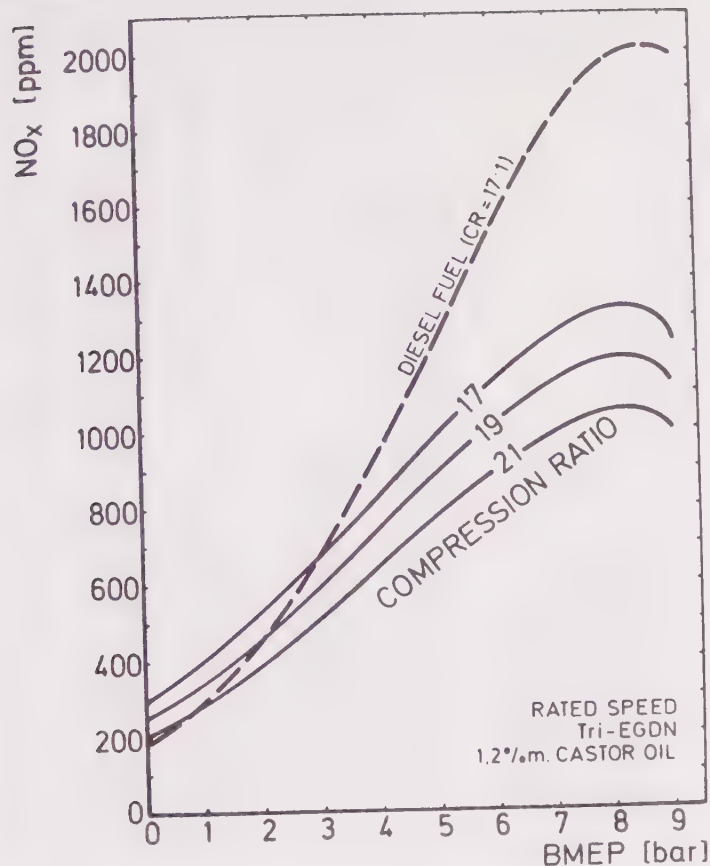


Fig. 12: Compression ratio and NO_x emission

sel fuel and 17:1 compression ratio. With a compression ratio of 17:1, the injection timing was the same with both fuels, whereas it was different for methanol operation with 21:1 compression ratio. The Tri-EGDN content of the methanol fuel was 3.5% by volume (6% by mass) with a compression ratio of 17:1, and 1.5% by volume (2.5% by mass) with a 21:1 compression ratio.

As expected, substantially reduced nitrogen oxide emissions were observed with methanol fuel. With a compression ratio of 17:1, NO_x emissions were only 68% of those during diesel fuel operation, and as low as only 50% with a 21:1 compression ratio. With retarded injection timing, a measure which could be applied due to the soot-free exhaust gas, a further reduction in NO_x emissions of about 10% could be achieved with only a small fuel consumption penalty.

Also represented in Fig. 15 are the relative emissions of carbon monoxide, which are of no practical importance compared to the federal CO emission standard. With a 17:1 compression ratio, the CO emission is significantly reduced with methanol fuel. This advantage is lost, however, when the compression ratio is increased to 21:1.

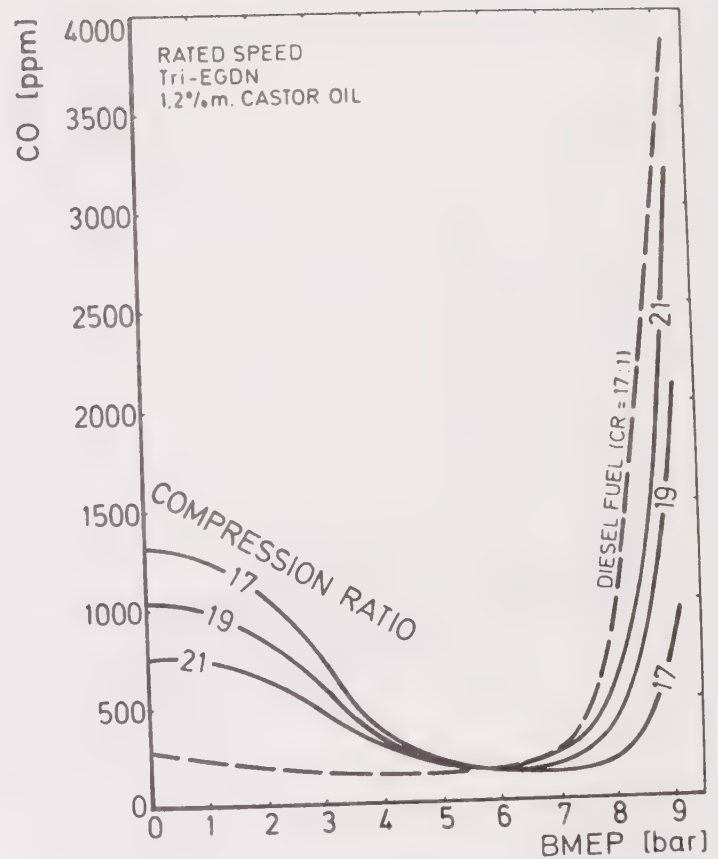


Fig. 13: Compression ratio and CO emission

The relative emission of unburnt hydrocarbons, however, is greatly increased with methanol fuel, as is also shown in Fig. 15. Due to the fairly low HC emission of the diesel-fuelled engine of about 0.5 g/BHP-h, however, the increase in hydrocarbon emissions with methanol fuel does not mean exceeding the respective standard of 1.3 g/BHP-h. Therefore, no oxidation catalyst is required. Once again, the application of a 21:1 compression ratio proves to be advantageous, it results in a lesser increase in the emission of unburnt hydrocarbons.

EMISSIONS OF UNBURNED METHANOL

Additional remarks need to be made concerning the hydrocarbon emission data given in Fig. 15. The calculation of the horsepower-related emissions from the HC concentrations in the exhaust gas measured during the 13-mode cycle is based upon certain pre-conditions. A theoretical fuel is assumed to be typical of diesel fuels, one which is composed solely of hydrogen and carbon with only one carbon atom per molecule and a molar H/C ratio of 1.85. It is also assumed that the unburnt hydrocarbons are equally composed.

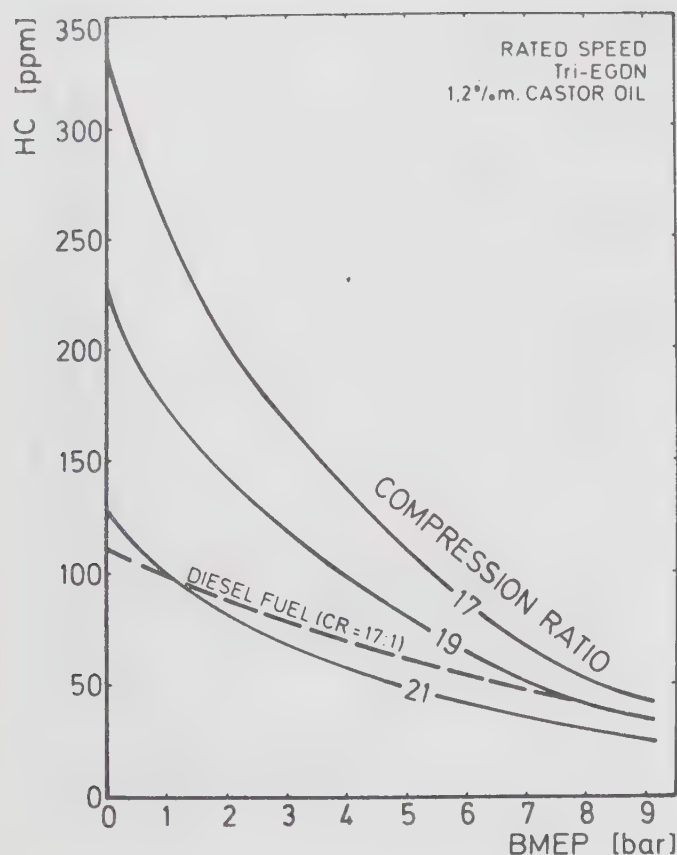


Fig. 14: Compression ratio and emission of unburnt hydrocarbons

As these pre-conditions do not apply in the case of methanol fuel, three different definitions of the relative HC emission of the methanol-fuelled engine are compared in Fig. 16. On the left, the HC emission results from Fig. 15 are reproduced. These were obtained from standard 13-mode cycle evaluation. They are expressed in terms of g/BHP-h emission of the aforementioned single-carbon atom diesel fuel with an H/C ratio of 1.85, related to g/BHP-h emissions of the same diesel fuel. With this definition, the HC emission results are higher than with diesel fuel and 17:1 compression ratio by 114% and 81% when methanol is used with 17:1 and 21:1 compression ratios, respectively.

The emission results presented in the center of Fig. 16 are obtained, when the differing H/C ratio and the oxygen content of methanol as well as the thereby caused increased exhaust gas mass are taken into account. Corresponding to the evaluation of 13-mode tests with diesel fuel, this assumes that the emission of unburnt hydrocarbons only takes place in the form of methanol. The results are appropriately expressed in terms of g/BHP-h emission of methanol, related to g/BHP-h emission of diesel fuel, and indicate

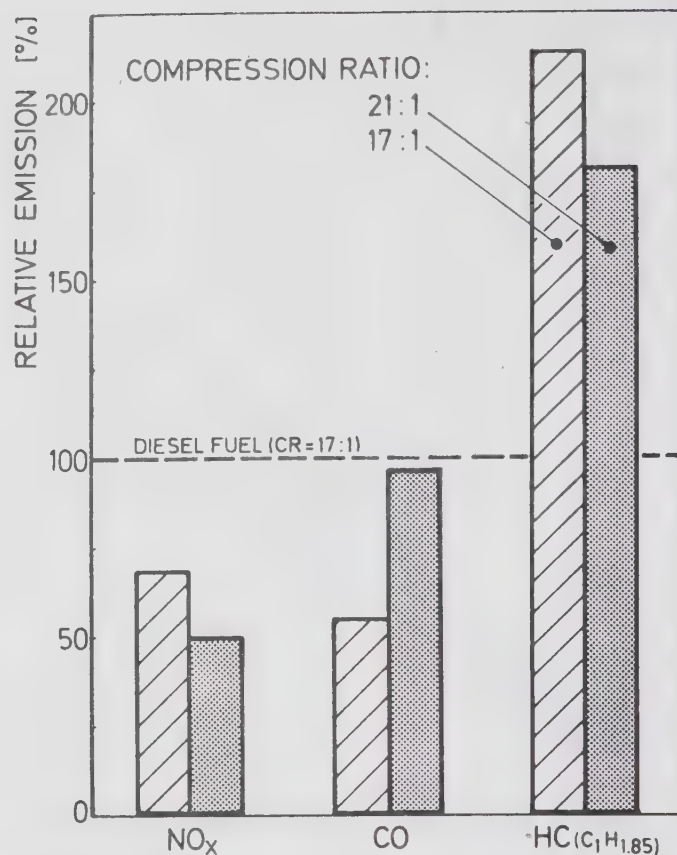


Fig. 15: Diesel fuel operation-related 13-mode cycle emission results with ignition-improved methanol

that the emission of unburnt methanol is 394% (CR = 17:1) and 318% (CR = 21:1) higher than the emission of diesel fuel with 17:1 compression ratio.

For the sake of completeness, the diesel fuel emission-related emission of unburnt methanol is shown also on a molar basis, on the right. With this definition, the emitted number of moles of unburnt methanol is 131% (CR = 17:1) and 69% (CR = 21:1) higher than the number of moles of the aforementioned diesel fuel emitted with 17:1 compression ratio.

It is not the purpose of this paper to recommend whether the emission of unburnt methanol should be compared on a mass or a molar basis to the emission of diesel-fuelled engines, when the application of methanol as a fuel for heavy-duty engines is considered from an environmental point of view. But, it should be understood that independent of the HC emission definition used, methanol-fuelled heavy-duty engines exhibit a considerably increased HC emissions, compared to diesel-fuelled engines, unless of course an oxidation catalyst is used.

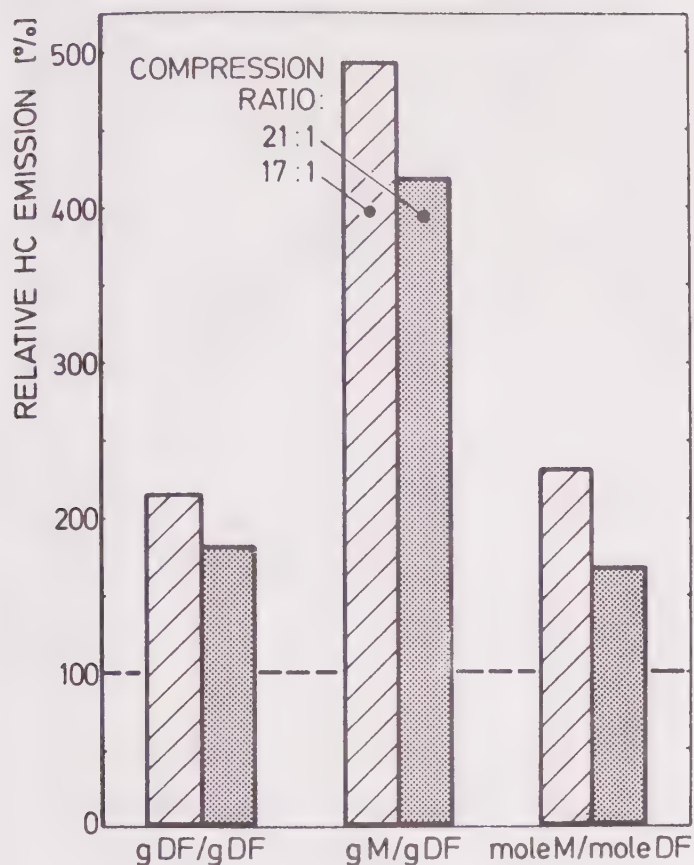


Fig. 16: Definition dependence of relative HC emission

ECONOMIC CONSIDERATIONS

When assessing the economics of heavy-duty diesel engine operation with ignition-improved methanol, several factors have to be taken into consideration:

- Further development work must be carried out on the engines, and new injection pumps for larger delivery volumes have to be designed. These development costs as well as those for the development of more suitable lubricating oils (14) and lubricity additives (10) have to be taken into account.
- Methanol fuel must be produced, stored and distributed separately from diesel fuel, which means that a costly infrastructure is required.
- The costs of methanol fuel, which is determined to a great extent by the required ignition improver content is decisive when evaluating the competitiveness with diesel fuel.

Fig. 17 shows the volumetric diesel fuel-related costs of ignition improver and of methanol below which economical operation is possible. If no ignition improver was required, the volumetric methanol/diesel fuel cost ratio would have to be below 44% to permit economic operation, due to the fact that 2.23 times as

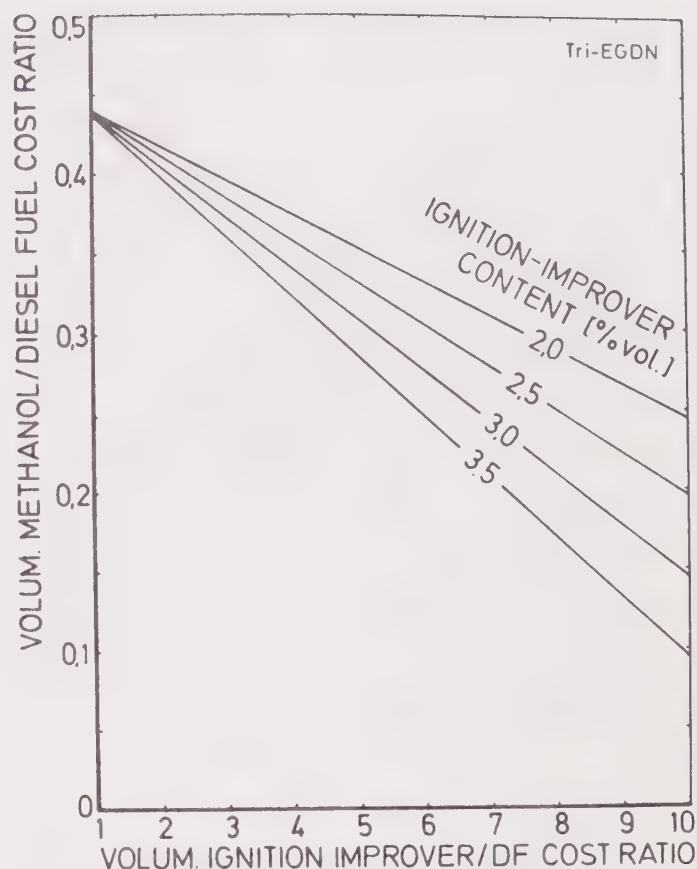


Fig. 17: Ignition improver percentage and competitive methanol cost

much methanol is required.

This ratio drops as the ignition improver content and costs increase. As shown before, increasing the compression ratio, e.g. from 17:1 to 21:1, results in a reduction of the ignition improver demand, e.g. from 3.5% by volume (6% by mass) to approximately 1.5% by volume (2.5% by mass). However, as can also be seen from Fig. 17, remarkable improvements in economy are only possible with expensive ignition improvers.

These considerations apply only under the condition of fuel-independent engine efficiencies. As was shown in Fig. 8, these are up to 5% higher with ignition-improved methanol. But, as can be seen from Fig. 18, even such considerable efficiency improvements affect the competitive fuel cost ratio only slightly.

The percentage reduction of Tri-EGDN demand with increasing compression ratio is shown in Fig. 19, relative to the requirement ascertained for a compression ratio of 17:1 (10). There is an even slightly greater saving, referring the necessary ignition improver percentage to the substituted diesel fuel volume. This is due to the fact that the heat value of methanol fuel rises with decreasing ignition improver content, as Tri-EGDN has a



Fig. 18: Fuel efficiency ratio and competitive methanol cost

lower heat value than methanol.

The competitiveness of ignition-improved methanol for use in compression ignition engines, compared to the application of neat methanol in spark ignition engines, is decisively governed by the necessary additive package, and particularly by the ignition improver cost. As the energy consumption of a heavy-duty diesel engine fuelled with ignition-improved methanol is about 20% less than of a comparable spark ignition engine (4), the costs of methanol with additives must not exceed the costs of neat methanol by more than the same percentage to remain competitive. However, as the costs of the various fuel components are not known and cannot be reliably assessed, the following considerations are based on relative, i.e. neat methanol-related, Tri-EGDN costs, both on a mass basis.

Fig. 20 gives the resultant added costs as a function of the compression ratio for Tri-EGDN costs of 2 to 7 times those of methanol. With a cost increase rate of 20% per unit of energy and 17:1 compression ratio, the cost of the ignition improver may be 4 times the cost of methanol. The limits of competitiveness with a 21:1 compression ratio would only be reached with a 7:1 Tri-EGDN/methanol

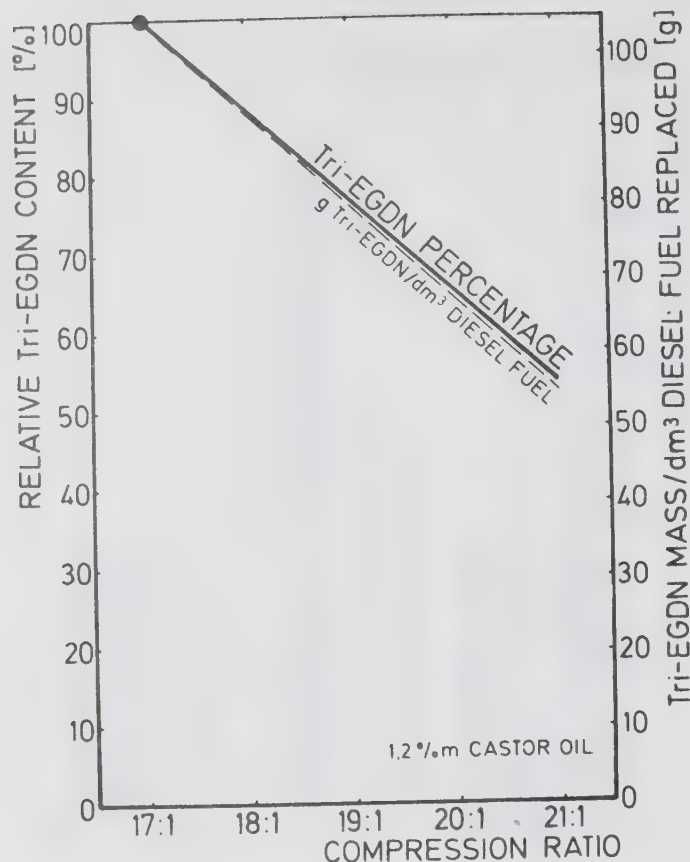


Fig. 19: Ignition improver savings with increased compression ratios

cost relation.

CONCLUSION

Methanol with an additive package, primarily containing an ignition improver, is technically suitable as an alternative fuel for heavy-duty diesel engines. Compared with diesel fuel operation, it not only offers advantages in terms of engine efficiency but in particular with regard to exhaust emissions.

It appears that the fuel-related mechanical problems described in a companion report (14) can be solved by modifying engine components and materials. Nevertheless, considerable research and development efforts will still be required.

So far, it cannot be assessed whether ignition-improved methanol will become viable from an economical and logistic point of view.

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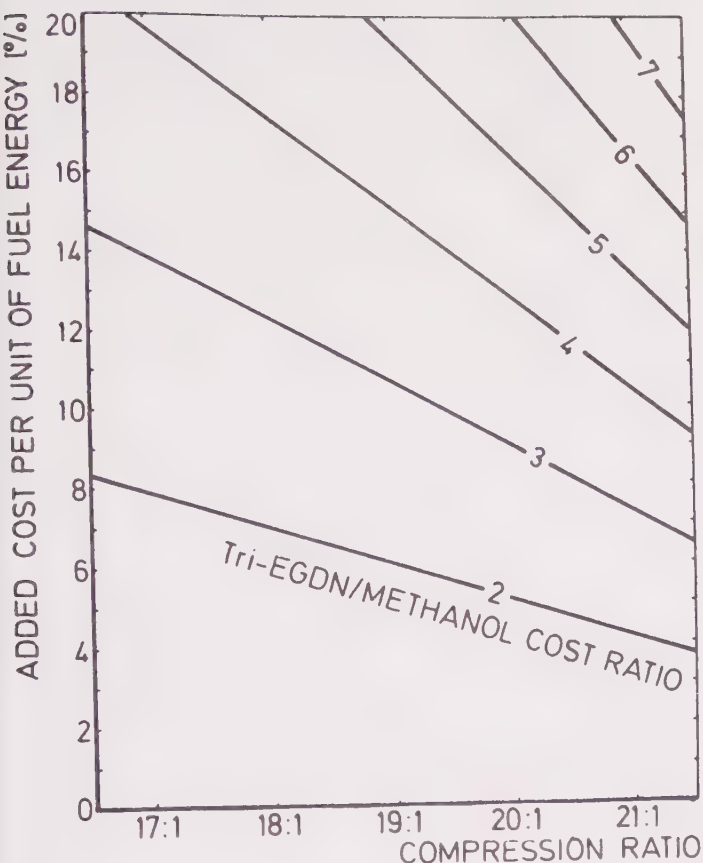


Fig. 20: Compression ratio, mass-related Tri-EGDN/methanol cost and fuel energy-related added cost

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SECTION III
SAFETY, ENVIRONMENTAL, AND ECONOMIC RELATED IMPACTS

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Safety Related Additives for Methanol Fuel

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ABSTRACT

This paper reviews the use of additives to improve safety aspects associated with the use of methanol as a motor fuel. A survey of the literature was conducted to determine candidate additives for methanol that produce one or more of the following properties: provide a visible or luminous flame, reduce the potential for skin contact, give a foul or unpleasant taste and odor, and act as an emetic. Candidate additives were reviewed to determine potential effectiveness, cost, ease of production, health problems, and effects on vehicle performance. Potential additives include complex hydrocarbon mixtures such as gasoline, alcohol soluble dyes and unpalatable compounds such as denatonium benzoate.

METHANOL is the simplest of alcohols. Synonyms include methyl alcohol, wood alcohol, wood spirit, carbinol, methylated spirit, methylol, monohydroxymethane, and methyl hydroxide. Methanol is a flammable, toxic liquid which burns with a non-luminous, bluish flame. It has a slight odor when pure, but the crude material may have a repulsive, pungent odor when produced from the destructive distillation of wood. It is miscible with water, ethanol, ether, benzene, ketones and most other organic solvents, and forms azeotropes with a number of compounds.

Methanol can be produced from raw material sources that can be partially oxidized under controlled situations to produce CO and H₂ (synthesis gases). The sources of methanol include non-renewable sources, such as municipal, agricultural, animal, and wood waste. The destructive distillation of wood as a method of production for methanol is no longer used in the United States, although some methanol is made commercially as a by-product in the production of charcoal. The conversion of

natural gas to methanol is currently the major source. Gasification of coal and the pyrolysis of waste, however, are considered to be potential raw material sources for large increases in future production of methanol.

During the nineteenth century methanol was widely used as a fuel for heating, lighting, and cooking, until it was replaced by kerosene. Since that time, methanol has been used as a fuel in engines, ranging from aircraft to racing vehicles. Recently, a major interest in methanol as a fuel has resulted from the potential reduction of exhaust emissions, independence from foreign oil supplies, and utilization of domestic surpluses of biomass.(1)*

Prior to 1880, the only commercial source of methanol was from the destructive distillation of wood and poisoning from methanol was virtually unknown. Wood alcohol, had a vile taste and disgusting odor and was rarely ingested. In the 1880's, a relatively inexpensive method of deodorizing wood alcohol was introduced and the resulting products were advertised as substitutes for ethanol. Not until 1923, when a group of dock workers in Germany were poisoned by ingestion, did the public finally realize that methanol really was a poison. During World War II, it was estimated that about six percent of the nonfatal cases of blindness to servicemen were caused by methanol.(2)

There has been a renewed concern over the toxic properties of methanol. The purpose of this paper is to identify additives for potential use in improving the safety aspects of methanol used as a motor fuel. This purpose was achieved through a survey of the literature and from contact with numerous individuals having experience related to methanol, fuel in general, specific types of additives, and health and safety. This paper is limited by the fact that few comprehensive studies have been conducted on types of additives examined. Candidate additives for methanol were evaluated regarding their ability to

*Numbers in parentheses designate references at end of paper

1) insure methanol burns with a visible flame, 2) prevent improper use of the fuel as a degreaser or cleaning agent, 3) give the fuel an unpleasant taste causing expectoration of methanol from one's mouth, 4) act as an emetic, and 5) produce an easily detected odor. The additives were evaluated as to effectiveness, cost, ease of production, possible health problems associated with the additive, and effects on vehicle performance.

FLAME LUMINOSITY

Neat methanol burns with a low luminosity bluish flame which is essentially invisible in daylight. Low flame luminosity is undesirable because an invisible fire is difficult to detect and more difficult to extinguish. A visible flame increases the safety factor since one could more easily detect any accidental fires and move to safety or take steps to extinguish the flame. Once located, a methanol fire can be easily extinguished with water because of solubility of methanol in water.

The luminosity of a burning substance is in part related to the formation of submicroscopic soot particles during the combustion process. These carbonaceous particles are heated by the flame and subsequently emit "grey-body" radiation at visible light wavelengths. Methanol burns with a cooler flame, and its combustion produces no carbonaceous particles.(3) Therefore, neat methanol has lower flame luminosity than most organic compounds.

There is no accepted standard for a visible flame. Most hydrocarbons function as methanol flame luminosity enhancers; however, concentrations of greater than 10 volume percent may be required to produce the desired effect. Addition of 10 volume percent gasoline in methanol has been reported to give the blend a flame luminosity equal to that of pure ethanol.(3)

In addition to enhancing the flame luminosity, a desirable characteristic of an additive would be persistence of the visible flame until all of the liquid is consumed or the fire is extinguished. Any additive that does not provide a luminous flame for the entire length of the burn introduces the risk that the observer could be misled into believing that the flame is gone when the color disappears. Several investigators have examined the time-dependent, flame luminosity for a number of hydrocarbons mixed with methanol. These data are included as Appendix Table 1 of this report.

The time-dependent behavior exhibited by methanol mixed with hydrocarbons can be readily understood on the basis of the methanol/additive distillation curves, the concentration of the additive, and the volatility of the additive.(4) Methanol forms minimum boiling azeotropes with many of the lower-molecular weight hydrocarbons. Upon burning of a pool of liquid, the additive is selectively distilled from the liquid phase, which concentrates the methanol. After sufficient quantity of the additive has been removed during the burning, the methanol dominates the liquid and the visible flame disappears. A comparison of high and low volatility additives demonstrates the effect on the flame luminosity. A high volatility, low boiling additive, such as

isopentane, produces a short-duration, intensely-luminous flame. Once the isopentane burns away, the remainder of the flame is indistinguishable from a flame of burning neat methanol. An additive with a higher boiling point such as o-xylene, has a low luminosity initially and becomes more luminous following selective distillation of the methanol from the liquid phase.

A flame luminosity study involving the addition of toluene, gasoline, and reformat (an aromatic gasoline blending stock) to methanol/pentane blends found toluene to be the most effective in increasing flame luminosity.(5) In another study, 4 volume percent hydroformate (reformat fraction distilling in the range of 55-220°C) blend produced a luminous flame throughout the entire burn, while a concentration of 10 volume percent unleaded gasoline was required to give a visible flame for the entire burn.(6) Individual components such as isopentane, toluene, xylene, and lower concentrations of unleaded gasoline, were not as effective in enhancing the flame luminosity.

The addition of water to methanol-dimethyl ether blends with toluene hydroformate, and LCN (light cut naphtha, catalytic cracker fraction distilling in the range of 30-165°C) was found to decrease flame luminosity.(6) This study suggested that the blends should contain less than 5 volume percent water to retain flame luminosity for the entire burn time.

The addition of organometallic compounds and/or metal organic salts to methanol is another means of increasing the flame visibility of methanol. Sodium acetate, a metal-organic salt used in Sterno (a commercially available methanol containing camping fuel), has been examined as a flame luminosity enhancer for methanol fuel. With sodium acetate, as the flame burns, the metal organic salt is concentrated in the liquid phase by selective distillation of methanol, and the salt precipitates out when the solubility product is exceeded.(4) Another organometallic compound, tetraethyllead, has been used in leaded gasoline for a number of years. It appears unlikely that organometallic compounds would produce the desired effect at concentrations that would be reasonable in motor fuel. In addition, many metals are detrimental to the operation of engines and exhaust systems of modern catalyst-equipped vehicles.

Alkyl borates and boron esters have also been proposed as a means of enhancing the flame luminosity of methanol.(7) Borates, which burn with a yellowish green flame when combined with alcohol, have been suggested as water scavengers in 85% methanol blends.(8) The suggested concentrations of borates, between 0.5 and 1.5 weight percent, however, are not expected to enhance the flame luminosity of methanol.

For enhancing flame luminosity, 10 to 15 percent volume percent of unleaded gasoline appears as a possible candidate. Large quantities of unleaded gasoline could readily be made available for mixing with methanol, and the necessity of building additional facilities to produce a special methanol additive would be avoided. There would be some increase in the cost of methanol with the addition of unleaded gasoline, depending on the amount of unleaded gasoline added (10 to 15 percent). This cost increase, however, would

be offset by the higher energy content of gasoline, approximately twice that of methanol. A summary of the additive costs are shown in Appendix Table 2.

The addition of complex mixtures of hydrocarbons, such as unleaded gasoline, to increase the flame luminosity of methanol, also aids in other safety-related areas. One of these areas is the flammability of the methanol vapor in a closed system. The flammability limit for methanol in air ranges from about 36 volume percent as an upper limit, to about 6 volume percent as a lower limit. By comparison, gasoline has a much narrower flammability range, with an upper limit of 7.6 percent and a lower limit of 1.4 percent. A methanol fuel blend with 20 volume percent gasoline has been found to be too rich in hydrocarbon vapors to ignite above an ambient temperature of -10°C .(5) The addition of 10 percent or more unleaded gasoline or other hydrocarbon mixtures would therefore be expected to reduce the possibility of an explosion in the vapor space of a closed container, such as a vehicle fuel tank.

Another problem associated with methanol fuel that would be aided by the addition of a complex hydrocarbon mixture, specially low molecular weight hydrocarbon, is the vehicle cold-start ability (i.e., the ability to start an engine at low ambient temperatures). The problem is related to methanol's relatively high latent heat of vaporization (506 BTU/lb as compared to 150 BTU/lb for gasoline). The addition of 12 volume percent gasoline to methanol has been shown to give a vehicle adequate cold startability at temperatures down to -10°C .(5)

The addition of complex mixtures of hydrocarbons such as gasoline to methanol fuels would also be expected to increase the odor intensity of the fuel blend and when mixed with water give an unattractive milky mixture with a strong odor of gasoline.(5) On the negative side, the addition of hydrocarbon mixtures to methanol fuel would be expected to increase the evaporative/refueling emissions throughout the fuel supply system.(15) Hydrocarbon emissions are also thought to be more reactive in the atmosphere than methanol.(16,17,18) Thus, the addition of HC mixtures to methanol would contribute more to photochemical smog formation than neat methanol fuel.

The addition of gasoline or a hydrocarbon mixture to methanol would produce some changes in the engine-out emissions of hydrocarbons, carbon monoxide, and oxides of nitrogen, however current vehicle emission control systems should minimize these differences. Also sulfur and aromatic-related exhaust emissions (sulfur dioxide, benzene, etc.) would increase as a function of their concentration in the additive.

TASTE DETERRENTS

Of the three major routes of methanol poisoning (ingestion, inhalation and absorption through the skin), direct ingestion is the quickest. Methanol does not have a strong or distinctive taste to identify its presence and has been mistaken for ethanol innumerable times. The addition of a bitter or foul tasting substance would help to deter

accidental poisoning and possibly even prevent some intentional ingestion of methanol fuel.

Researchers have stated that the usual fatal dose by direct ingestion of methanol is between 50 and 100 ml (2-4 oz.), although 25-50 (1-2 oz.) has often been fatal if not treated immediately.(1,2) The lowest reported fatal dose was 3 teaspoons (about 15 ml) of 40% methanol (approximately 6 ml pure methanol) and the highest dose of a survivor was one pint (500 ml) of the same mixture (approximately 200 ml of pure methanol). It has been stated, however, that the chance of a 6 ml dose causing death is exceedingly low.(19) The consumption of ethanol prior to or in conjunction with methanol has been found to decrease the toxic effect. Ethanol has been shown to compete effectively for the enzyme responsible for the conversion of methanol to formaldehyde and formic acid. The production of formaldehyde and formic acid is suspected of being responsible for some of the toxic effects of methanol.

Although little information exists on the addition of materials to methanol to make it undrinkable, denaturants have been added to commercial ethanol to render it unfit for human consumption for over 60 years. Although denaturants are most commonly involved with ethanol, some of these substances could be used in methanol as a taste deterrent. Most of the formulas for denaturing ethanol to be used as a fuel contain methanol and/or gasoline. A list of several important factors involved in choosing a suitable denaturant for alcohol fuels includes(20):

- Should match the thermal and physical properties of the alcohol
- Should add to the energy content
- Should impart a disagreeable taste or smell
- Should not be capable of being easily eliminated
- Should be capable of being easily and reliably detected
- Should not add to the emission levels
- Should not leave any objectionable residue
- Should be readily available
- Should not add appreciably to the cost
- Should not complicate the regulatory compliance

Researchers have stated that the toxicity of a blend of gasoline and methanol is only slightly more of a hazard than ordinary gasoline.(5) This is due to the prominent gasoline odor in the blend and the difficulty in separating methanol from the blend by "casual" means to be pure enough to be consumable. The addition of water to a gasoline (5 or more volume percent) and methanol blend was found to produce an unattractive milky mixture. Lower concentrations of gasoline were found to be not as objectionable,

although the gasoline odor was still noticeable.

Several companies have expressed interest in producing blends of methanol and methylal (dimethoxymethane), an additive to increase the engine cold starting properties.(21,22) Although no claims have been made concerning the taste of methylal/methanol blends, methylal should act as a deterrent to the ingestion of methanol because of its pungent taste and chloroform-like odor.

There are a number of individual compounds that can be added to give methanol a highly bitter and foul taste, however they may need to be used in conjunction with other additives because they may be selectively removed from methanol by distillation. Denatonium benzoate, currently used in paints, detergents and many other household materials, would probably be an effective additive, due to its relatively low bitterness threshold and corresponding low cost (See Appendix Table 2). A number of other compounds have been proposed and used as additives to produce a foul- or bitter-tasting product. Several of these compounds are listed in Table 1, along with their bitterness thresholds (if available), which are indications of the minimum concentration required to produce a bitter taste.

DYES AND COLORANTS

Dermal contact with methanol has been shown to be one of the routes in methanol poisoning. The addition of a dye would help to deter the improper use of methanol as a degreaser or cleaning agent, during which skin contact and subsequent absorption could result. If a dye temporarily colored the skin, one would probably not use the treated methanol as a cleaning solvent. An intense or repulsive color would also tend to deter the ingestion of methanol.

The cutaneous exposure of human subjects to methanol has been examined by a number of investigators. A mean value for the absorption rate of methanol through the skin has been determined to be $0.192 \text{ mg/cm}^2/\text{min}$.(24) Using this value, researchers have calculated the absorption from immersing the whole hand (440 cm^2 surface area) in methanol for twenty minutes. This immersion would result in the absorption of 1.7 mg (2 ml) of methanol. The absorption of 2 ml of methanol is approximately 5 percent of the usual fatal dose, and 33 percent of the smallest reported fatal dose (6 ml) by ingestion. Although it is unlikely that one would immerse a hand in methanol for twenty minutes without some

TABLE 1. COMPOUNDS FOR FOUL TASTE(23)

Compound	Bitterness Threshold	Comments
Benzyl alcohol		sharp burning taste; faint aromatic odor
Denatonium benzoate	1:1,000,000	most bitter substance known to man. Added to toxic substances as a deterrent to accidental ingestion
Brucine	1:220,000	very bitter taste; very poisonous; used in denaturing alcohols and oils
Dimethylformamide		universal organic solvent; faint amine odor
Methylal		pungent taste; chloroform odor; volatile flammable liquid
pyridine		sharp taste; characteristic disagreeable odor; flammable, colorless liquid
Quassin	1:60,000	one of the bitter constituents of the wood of <i>Quassia amara</i> L; very bitter
Quinine	1:30,000 (quinine hydrochloride)	most important alkaloid of cinchona bark; salts also bitter
Strychnine	1:130,000	very bitter; extremely poisonous
Sucrose octaacetate		intensely bitter; hygroscopic; denaturant for alcohol (ethanol)

discomfort, case studies have shown that industrial workers (painters, varnishers, hatters, etc.) have experienced blindness or even death from cutaneous exposure to methanol.

The rate of methanol absorption has also been found to be time dependent and consist of two phases(24) (Figure 1). In the first phase, the absorption rate increased with longer times until about 30 minutes has elapsed. The increase in absorption rate for the first phase is approximately $0.0053 \text{ mg/cm}^2/\text{min}$. The second phase shows a slight decrease in absorption rate for the remainder of the exposure time (no data available for exposures longer than 60 minutes). The overall absorption rate of methanol is considered comparable to those of benzene, xylene, and carbon disulfide.

Blends of 85, 50, and 15 percent gasoline with methanol have been used to investigate the influence of methanol-gasoline combinations on dermal absorption.(25) The effectiveness of barrier creams for preventing methanol absorption have also been investigated. Methanol alone was found to cause less dermal change than methanol/gasoline blends which gave the skin a very white and dry appearance. The methanol/gasoline blends were described by test subjects as irritating, with the most discomfort caused by the 50 percent gasoline mixture. The gasoline mixtures modified the absorption process and allowed greater amounts of methanol to be absorbed. The use of barrier creams failed to protect the skin from methanol penetration, and appeared to reduce the capacity of normal skin to resist the absorption of methanol. According to the researchers in this study, the absorption of methanol from a mixture with gasoline was more likely to be irritating because of the de-fatting effect caused by the petroleum distillate.

The addition of dyes has historically been used to identify a variety of fluids used in automotive applications; however, the use of a dye to stain the skin is not a common practice. A variety of alcohol-soluble dyes are commercially available in a rainbow of colors. Commercial methanol products have typically been blue in color.

The concentration of dye in methanol needed to stain the skin is considerably higher than the concentration of dye needed to give a distinctive color to methanol. Five methanol-soluble dyes, including a blue, a yellow, an orange, and two shades of red, were found to give discernable colors at a concentration of 4 mg per gallon. Concentrations on the order of 400 mg of dye per gallon were needed to cause staining of the skin, and only the blue dye caused any significant discoloration. The use of blue dye as a colorant would conform to the coloration used in commercial products. For comparison, the dyes used in gasoline are present in concentrations up to 40 mg per gallon. A methanol fuel containing a dye to stain the skin could result in irreversible coloration of clothing and vehicle components, and lead to additional exposures when trying to remove the dye from skin or clothing.

The use of dyes in methanol would probably have little or no impact on the exhaust emissions, if the dyes were used at concentrations equal to or lower than those currently used in gasoline (up to 40

mg per gallon). The impact on the exhaust emissions from higher dye concentrations would depend on the chemical makeup of the dye. Dyes containing metals such as manganese (permanganate) or halogens (chlorine, bromine, iodine) should be avoided because they may damage the emission control devices.

EMETIC

The use of emetics as additives to methanol is another means of reducing the severity of poisoning due to its ingestion. As emetic is an agent or substance that induces vomiting. Another related group of compounds, "antialcoholics," is also a possible means of deterring the consumption of methanol.

The usefulness of emetics generally occurs after a poison has been ingested, rather than acting as a deterrent. The addition of an emetic should be present in high enough concentrations to induce vomiting whenever a harmful amount of methanol has been swallowed, should be reliable, and should act in a very short period of time to minimize the absorption by the body. Common practices to induce vomiting include the administration of 10 g of table salt in 20 ml of warm water (2 teaspoons in a glass of warm water)(26), the administration of 2 teaspoons of sodium bicarbonate (baking soda) in a glass of water(23), or the administration of 30 ml (1 oz) of syrup of ipecac to the victim.(27) Table salt and sodium bicarbonate are not considered viable additives for methanol fuel.

Ipecac, the emetic generally recognized by the medical profession, is obtained from the dried rhizome and root of a plant found in Central and South America. Ipecac consists of a variety of alkaloids, including emetine, cephaeline, emetamine, ipecacuanhic acid, psychotrine and methyl psychotrine. Emetine is one of the principal alkaloids of ipecac, and it is used in Great Britain as an emetic(28) and in third world countries as an antiamebic.

Antialcoholics are administered to individuals before ingestion of ethanol, and are used to treat chronic alcoholics as a means of reducing their dependency on ethanol. After the administration of antialcoholics such as disulfiram (tetramethylthiuram disulfide) or citrated calcium cyanamide, a patient experiences a number of physical responses, including vomiting, when ethanol is ingested. Since antialcoholics must be administered before the consumption of an alcohol, their use is impractical as an emetic additive to methanol.

Based on the large quantity of an emetic which would need to be added to methanol to produce the desired effect and the corresponding cost associated with this amount of emetic, the use of an emetic in methanol fuel does not appear to be practical. About 2 ml of pure ipecac extract or about 30 ml of the syrup of ipecac (ethanol base) are required to induce vomiting.(28) A dose of 25 ml of methanol had been reported as fatal in some cases if not treated immediately; therefore, 2 ml of ipecac extract would be required for each 25 ml of methanol fuel (8 percent). The cost of adding emetics to methanol is summarized in Appendix Table 2.

ODORANTS

Another major route of methanol poisoning is inhalation. Methanol does not have a strong or distinctive odor to identify its presence, and when pure, has such a low odor intensity that one could be exposed to hazardous levels without realizing it. The addition of odorants would help identify the presence of methanol, and serve as a warning of possible hazardous conditions. A suitable odorant would need to coevaporate with methanol and be detectable at a much lower concentration than methanol vapor alone.

The Occupational Safety and Health Administration (OSHA) has set a workplace ceiling level standard of 150 ppm (200 mg/m³) for methanol in air. The IDLH (Immediately Dangerous to Life or Health) value for methanol is set at 25,000 ppm, and the lower explosive limit is approximately 67,000 ppm. The values for the odor threshold have been reported to range from 100 to 1200 ppm.(19) Varying levels of impurities in methanol may account for this discrepancy. If the odor threshold is at the upper end of the range, then one might not be able to detect levels where serious physiological effects could result.

Researchers screened a number of substances for their effect on the odor threshold of methanol.(5) The most effective odorants tested in the study were determined to be n-butyl mercaptan and ethyl acrylate. Gasoline, giving an "old paint" odor to the blend, was not considered as effective as the others. At very low concentrations, the odor of ethyl acrylate was described as pleasant, while at higher concentrations, the odor was found to be extremely irritating. The two mercaptans screened in the study produced very distinctive odors even at very low concentrations, however, n-butyl mercaptan was considered more unpleasant than t-butyl mercaptan. One disadvantage of using mercaptans as methanol additives is possible confusion with a natural gas leak, since natural gas contains mercaptans as odorants. Organic sulfides (i.e., methyl sulfide, ethyl sulfide) have an odor slightly different from, but equivalent in intensity to that of the mercaptans. They are also more resistant to oxidation, and would be viable alternates for odorous additives to methanol.

Vehicles using methanol fuel with mercaptans or organic sulfides as odorant additives would produce exhaust emissions containing sulfur dioxide and sulfate. Using 200 milligrams of mercaptan per gallon of methanol would result in sulfur emissions on the order of one-tenth those from the sulfur in conventional gasoline.

The use of mercaptans or organic sulfides at the levels necessary to produce a strong and unpleasant odor in methanol should not provide any problems with vehicle operation or with the health of people using the fuel blend. The use of ethyl acrylate as an odorant may not be as practical as the sulfur-containing odorants, since ethyl acrylate levels that were high enough to be detected were also irritating to the eyes and mucous membranes. The cost of adding odorants is summarized in Appendix Table 2.

OTHER METHODS TO INCREASE SAFETY

In addition to chemical means for improving safety in the handling of methanol fuel, several additional methods have been proposed, including fuel labeling, antisiphoning methods, sealed fuel handling systems, and methanol education.

The National Institute for Occupational Safety and Health (NIOSH) has recommended standard labeling of methanol containers and of areas where methanol is present.(29) Methanol is regulated by the Federal Hazardous Substances Act and must be labeled with "Danger," "Poison," "Cannot be made nonpoisonous," and the skull and crossbones symbol. The statement of hazard must include the language, "Vapor Harmful." The additional warning "not be taken internally" has also been suggested.(30) All references to "alcohol" should be avoided in the distribution and discussion of methanol, since the lay person may identify the term alcohol with ethanol and deduce that it is drinkable. A term, such as "fuel methanol," would be preferable.

Antisiphoning devices or techniques have been suggested to remove the possibility of oral or dermal contact with fuel methanol.(31) Also suggested were sealed handling systems and redesigned fuel delivery systems. A closed system would help to eliminate dermal contact and exposure to methanol vapors. The racing organizations (CART and USAC) have been using a closed fueling system, called a "dry break" system, for many years on their racing vehicles. The system is composed of self-closing, spring-loaded valves which do not allow the fuel to flow until the union has been completed. This system, in conjunction with ground wires and fire proof clothing, helps to reduce exposure to methanol and the possibility of fire-related accidents.

Education of the general public to the dangers related to methanol would also be of great importance. Although this is probably the least cost effective method of safety enhancement, the frequency of serious accidental ingestion or exposure should be appreciably reduced. A NIOSH report concludes that the safe handling of methanol depends to a great extent upon the effectiveness of employee education, intelligent supervision, and the use of safe equipment.(29) Although education is not the complete answer, it would help to reduce many of the accidental hazards involved with methanol.

SUMMARY

The study, on which this paper is based, involved a review of methods, primarily additives, for improving the safety aspects of methanol fuel. Additives were evaluated regarding their ability to 1) insure that methanol burns with a visible flame, 2) reduce the potential for skin contact, 3) give the fuel an unpleasant taste causing expectoration of any methanol accidentally taken into the mouth, 4) act as an emetic, and 5) produce an easily detectable odor. Effectiveness, cost, ease of production, health problems associated with use, and estimated effects on vehicle performance were taken into account in the evaluations.

CONCLUSIONS

1. For flame luminosity enhancement, a practical and effective additive would be 10 to 15 percent of gasoline or other complex mixtures of hydrocarbons. These mixtures form a number of azeotropes with methanol which burn with a visible flame that persists throughout the duration of a fire. The addition of complex mixtures of hydrocarbons, such as unleaded gasoline, also aid in other related areas such as reducing the flammability of the methanol vapor in a closed system, increasing the ability to start an engine at low ambient temperatures, and increasing the odor and taste intensity of the fuel blend.

2. Gasoline also appears to be a reasonable additive to methanol as a taste deterrent. Gasoline can not be removed easily from a mixture with methanol, and researchers have reported that even a determined consumer would have difficulties in swallowing such a mixture. Denatonium benzoate, a taste deterrent used in paints, detergents, and other products is one of the most bitter substances known to man, and would also be a prime candidate as a taste deterrent for methanol.

3. A number of dyes in a variety of colors are soluble in methanol. Commercial methanol products have typically been blue in color, and a continuation of this practice would be logical for fuel methanol. A concentration of only 4 milligrams of dye per gallon of methanol has been found to give an easily recognizable color. A concentration of 400 milligrams per gallon, however, was needed to stain the skin. However, the desirability for the use of such large concentrations of dyes would have to be investigated, as a methanol fuel containing a sufficient dye to stain the skin could result in irreversible coloration of clothing and vehicle components, cause excess engine wear due to deposits, and to additional exposures when trying to remove the dye from skin or clothing.

4. The use of an emetic as an additive for methanol was evaluated to determine if its use would be practical to induce vomiting in the event the methanol fuel was consumed. This appears impractical, however, as the necessary concentration of an emetic (such as ipecac) in methanol was found to be about eight volume percent. The use of a large quantity of an emetic in fuel could also cause engine damage.

5. Due to low odor intensity of methanol, added odorants are necessary to assure the olfactory detection of methanol vapors. Mercaptans have been shown to be effective odorants for methanol at concentrations as low as 200 mg per gallon methanol. Mercaptans are used in natural gas, so their use in methanol could cause some confusion (i.e., is it a methanol spill or natural gas leak?) when their odor is detected. The more chemically stable organic sulfides, which have a slightly different but equally intense odor, may be more desirable.

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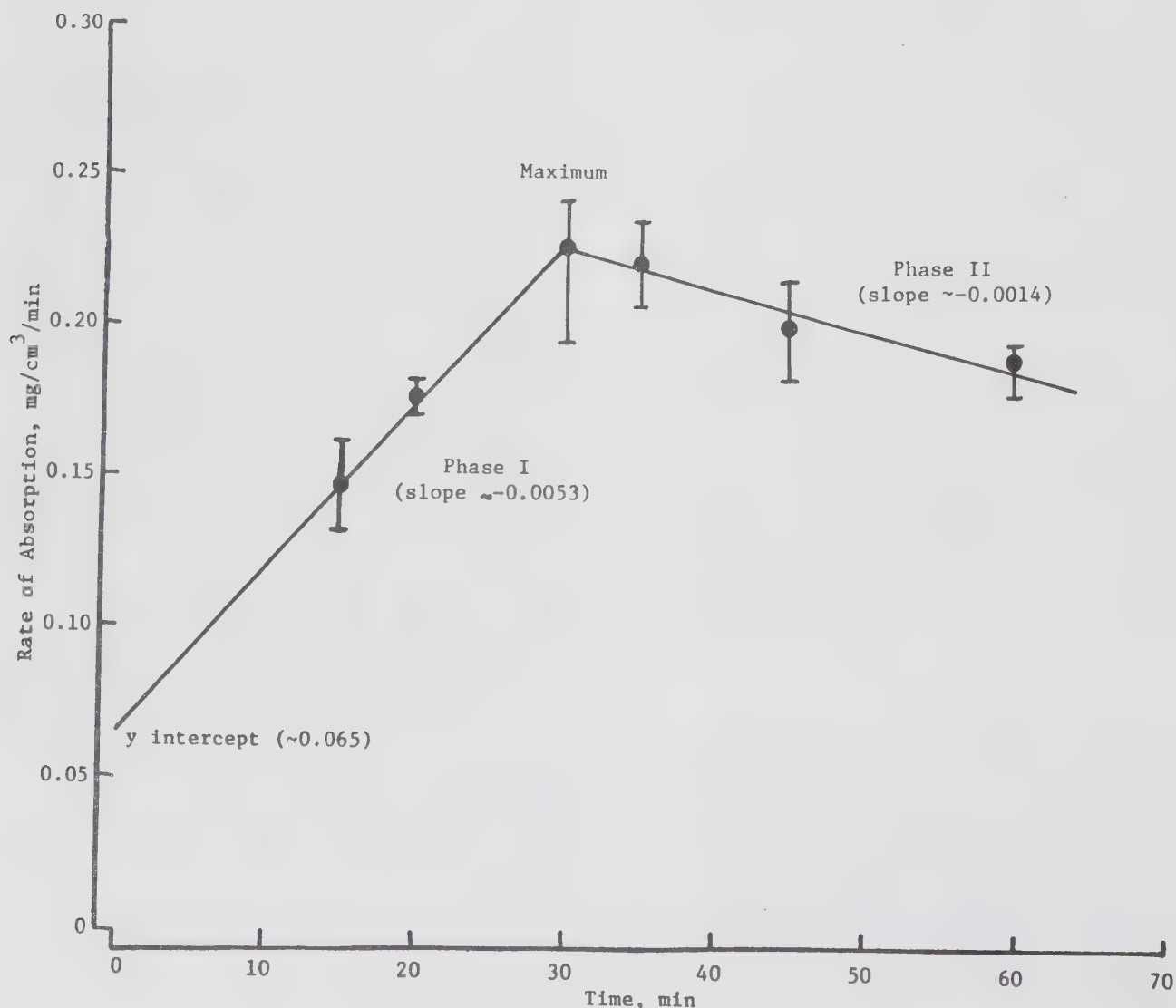


Fig. 1 - Experimental rate of absorption for methanol versus time(24)

APPENDIX TABLE 1. TIME DEPENDENT FLAME LUMINOSITY WITH METHANOL

Additive	Volume % of Additive	% Flame Luminosity Time ^a	Comments
methanol	--	0	Not visible except in dark ⁽⁴⁾
isopropyl alcohol	6	--	Light yellow flame barely visible ⁽⁴⁾
t-butanol	6	--	Light yellow flame barely visible ⁽⁴⁾
light hydrocarbons	5		Generally poor ⁽⁶⁾
light hydrocarbons	10		Substantially improved ⁽⁶⁾
DME ^b	5	0	(4)
isopentane	6	0	Few flashes of yellow ⁽⁴⁾
isopentane + DME	2 5	0	Few flashes ⁽⁴⁾
isopentane + toluene + DME	2 2 4.6	43	(4)
Hexane + DME	2 4	10	(4)
Cyclohexane	10	33	Flame luminosity decreased until it resembled burning methanol ⁽⁵⁾
toluene	2	0	Few flashes of yellow ⁽⁴⁾
toluene		90	Strong luminosity ⁽⁵⁾
toluene + pentane	2 5		Substantially increased flame luminosity and visibility ⁽⁶⁾
toluene + pentane	2 10		Substantially increased flame luminosity and visibility ⁽⁶⁾
toluene + pentane	5 5	50	(6)
toluene + pentane	5 10	70	(6)
toluene + DME	2 3.6	57	(4)
toluene + DME	2 5.4	83	Non-luminous in mid-period ⁽⁴⁾
toluene + DME	4 3.9	82	(4)
toluene + DME	4 5.6	90	(4)
toluene + DME	6 6	89	(4)

APPENDIX TABLE 1 (Cont'd). TIME DEPENDENT FLAME LUMINOSITY WITH METHANOL

Additive	Volume % of Additive	% Flame Luminosity Time ^a	Comments
toluene + LCN ^c	2 8	56	(4)
toluene + DME + LCN	2 4.5 2	73	(4)
toluene + DME + LCN	2 3.8 4	100	(4)
toluene + DME + LCN	2 4.9 4	100	(4)
toluene + DME + LCN	2 6 4	100	(4)
toluene + DME + xylene	1 6 1		(4)
toluene + DME + xylene	2 6 2	94	(4)
xylene	2	0	Few flashes of yellow ⁽⁴⁾
LCN + DME	2 5	0	Few flashes ⁽⁴⁾
LCN + DME	4 4	23	(4)
LCN + DME	6 5.7	43	(4)
LCN + DME + hydroformate ^d	2 4 2	42	(4)
hydroformate	4	100	Light yellow flame ⁽⁴⁾
hydroformate + DME	2 4.9	0	Few flashes ⁽⁴⁾
hydroformate + DME	4 3.8	100	(4)
hydroformate + DME	4 4.9	100	(4)
hydroformate + DME	4 5.7	100	(4)

APPENDIX TABLE 1 (Cont'd). TIME DEPENDENT FLAME LUMINOSITY WITH METHANOL

Additive	Volume % of Additive	% Flame Luminosity Time ^a	Comments
cat pentane ^e + DME	2 5	0	Few flashes ⁽⁴⁾
cat pentane + DME	4 4	0	Few flashes ⁽⁴⁾
unleaded gasoline (14.5 RVP)	6	50	Light yellow flame ⁽⁴⁾
unleaded gasoline (14.5 RVP)	10	100	(4)
gasoline	10		Equal to burning neat ethanol ⁽⁸⁾
gasoline	10	50	(6)
gasoline	15	100	(6)
gasoline + pentane	5 5	20	Visible flame ⁽⁶⁾
gasoline + pentane	5 10	30	Visible flame ⁽⁶⁾
reformate ^f + pentane	5 5	20	Visible flame ⁽⁶⁾
reformate + pentane	5 10	30	Visible flame ⁽⁶⁾

^aPortion of time that flame was yellow and clearly visible

^bDimethyl ether

^cLight Cat Naphtha-Catalytic cracker fraction distilling in the range of 30-165°C

^dReformate fraction distilling in range of 55-220°C

^eCatalytic cracker fraction distilling in the range of 25-150°C

^fAromatic gasoline blending stock

APPENDIX TABLE 2. SUMMARY OF ADDITIVE COSTS

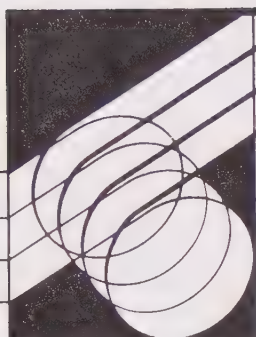
<u>Name</u>	<u>Cost Increase of Quantity</u>	<u>Methanol Fuel^a</u>	<u>Comments</u>
<u>Flame Luminosity</u>			
Gasoline	10 volume %	4¢/gal.	Based on average 1983 Gulf Coast refinery price for unleaded gasoline (84¢/gal)(10)
<u>Taste Deterrents</u>			
Denatonium benzoate	8 mg/gal.	2¢/gal.	Current market price-\$14/oz(11)
<u>Dyes and Colorants</u>			
Powdered commercial alcohol soluble dyes	400 mg/gal. 40 mg/gal.	2¢/gal. 0.2¢/gal.	Current average price of \$19/lb(12)
<u>Emetics</u>			
Ipecac	8 volume %	\$84/gal.	Current market price-\$120/lb(13)
<u>Odorants</u>			
Mercaptans and organic sulfides	0.2 g/gal.	0.05¢/gal.	Current market price range from \$1.04 to 1.08/gal.(14)

^aBased on 1983 Gulf Coast spot barge price for methanol of 44¢/gal.(9)

HEALTH EFFECTS INSTITUTE

H E I

SPECIAL REPORT



**Automotive Methanol Vapors and Human Health:
An Evaluation of Existing Scientific Information
And Issues for Future Research**

Report of the Institute's
Health Research Committee
May 1987

The Health Effects Institute (HEI) is a non-profit corporation founded in 1980 to assure that objective, credible, high-quality scientific studies are conducted on the potential human health effects of motor vehicle emissions.

Funded equally by the U.S. Environmental Protection Agency (EPA) and 26 automotive manufacturers or marketers in the United States, HEI is independently governed. Its research projects are selected, conducted, and evaluated according to a careful public process, including a rigorous peer review process, to assure both credibility and high scientific standards.

HEI makes no recommendations on regulatory and social policy. Its goal, as stated by former EPA Administrator William D. Ruckelshaus, is "simply to gain acceptance by all parties of the data that may be necessary for future regulations."

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STATEMENT FROM THE HEI BOARD OF DIRECTORS

The manufacturers of motor vehicles and the Environmental Protection Agency (EPA) have a responsibility under the Clean Air Act to ensure that any new technology affecting mobile source emissions will not pose an "unreasonable risk to the public health." (Section 202 (a) (4)). EPA has been requesting the Health Effects Institute (HEI) since 1983 to undertake a research program to determine what emissions-related health problems, if any, would emerge if methanol were to become more widely used as an automotive fuel.

Methanol-fueled vehicles emit both formaldehyde and methanol vapors. In 1985, HEI began to fund a research program to investigate the potential health effects of aldehydes, including formaldehyde. The HEI Health Research Committee, with our approval, decided to undertake additional analysis before proceeding with research on methanol vapors. This report contains the Committee's assessment. We are publishing it because we believe it will provide considerable guidance to government and industry, as policies relating to methanol fuel are considered.

The report focuses on the potential health effects of exposure of the general public to methanol vapor that might result from an introduction of methanol-powered motor vehicles in the general fleet. The report also evaluates, insofar as present knowledge permits, the likely health implications of such exposure. The report excludes analysis of the effects of accidental spills, ingestion, and worker exposure.

We have examined the report carefully, and we believe it represents a responsible summary of the current state of knowledge about the effects of methanol and the likely impact of exposure to its vapors. The Health Research Committee's analysis of the available evidence indicates that chronic exposure of people to low levels of methanol emissions is not likely to trigger known mechanisms of methanol's toxicity.

There has not been, however, sufficient research to eliminate entirely the possibility that health effects could occur at low levels of chronic exposure to methanol. A study of non-human primates chronically exposed to methanol vapor at moderate to high concentrations was recently completed in Japan. The summary report of that study, although rather sketchy, indicates the possibility of biological effects at exposure levels toward the upper end of the range of levels that have been predicted to arise from vehicular emissions. Accordingly, the specific findings of this study must be obtained, clarified if possible — a difficult and time-consuming task — and the research pressed further, if necessary. This appears particularly important if one goes beyond the foreseeable future and

contemplates the immense scale of methanol use that would result if methanol were to become a dominant fuel in the next century.

On balance, we believe that, given the anticipated uses of methanol as a motor vehicle fuel in the foreseeable future, the weight of available scientific evidence indicates that exposure to methanol vapors is not likely to cause adverse health effects. Health concerns regarding methanol vapor should not prevent government and industry from encouraging the development and use of methanol fuels, assuming that such development and use are otherwise in the public interest.

The Health Effects Institute and other research organizations are continuing to investigate the potential health effects from increased formaldehyde emissions that may result from methanol's use. The results of those inquiries will become available over the next several years. We believe that prudent public policy suggests that an additional modest research investment be made by appropriate research institutions, and perhaps by HEI, to reduce uncertainties further in estimating the health risks of low-level exposures to methanol, and to enhance the public's confidence in methanol technology.

Problems at relatively minor usage levels might only become evident as billions of gallons are introduced annually. It seems wise to ensure now that the possibility of adverse health consequences is minimized. It is in this light that any further research is prudent. But our best current assessment is that methanol fuel, under intended conditions of use, does not pose an unreasonable risk to the public health attributable to emission of methanol vapors from the tailpipe of motor vehicles.

In addition to thanking the entire HEI Health Research Committee for its efforts in shaping this document, we would particularly like to thank Dr. Walter Rosenblith, Chairman of the Committee, who directed this effort, Dr. Robert Kavet, who was the primary author of this report when he served as Senior Staff Scientist at HEI, and Dr. Roger McClellan, who gave generously of his time to ensure the quality of this effort.

*Archibald Cox, Chairman
William O. Baker
Donald Kennedy
Charles W. Powers*

May, 1987

AUTOMOTIVE METHANOL VAPORS AND HUMAN HEALTH:

An Evaluation of Existing Scientific Information And Issues for Future Research

EXECUTIVE SUMMARY

Methanol has the potential to become a major automotive fuel in the United States in the next century. One attractive feature linked to methanol's use is that emissions from methanol-fueled vehicles are expected to result in ambient concentrations of criteria pollutants that are no greater than and, quite likely, lower than those that result from gasoline or diesel emissions. However, the introduction of methanol also may result in increased exposure of the public to methanol and formaldehyde, both currently unregulated. The Environmental Protection Agency (EPA) has identified the importance of technically evaluating these relevant health issues. The Health Effects Institute (HEI) shares the EPA's concern and already has initiated laboratory research to investigate the health effects of aldehydes.

This report, prepared by HEI at the EPA's request, evaluates specifically the health consequences to humans that may result from inhalation of methanol vapors either emitted from methanol-fueled vehicles or during self-service refueling. The report's objectives are (1) to review the nature and mechanisms of methanol's toxicity, (2) to evaluate whether or not methanol's known effects might be expected at the anticipated low levels of intermittent exposure associated with increased use of methanol as a vehicle fuel, and (3) to identify both the areas in which critical knowledge is lacking and the research that could supply the needed information.

Anticipated Exposure Levels of Methanol

Data that estimate the range of potential exposure concentrations of methanol are essential to establish whether or not particular biologic or health effects are likely to occur. The EPA has conducted studies that project concentrations of methanol that will occur in a variety of scenarios. These include (1) three traffic situations - street canyon, roadway tunnel, and expressway; (2) exposures in both public parking and personal garages; (3) and exposures during self-service refueling. The analyses take into account both the driving conditions and the vehicle operating mode, as well as the contribution to emissions of vehicles that are not operating according to certification standards.

The highest exposures are expected in the garage scenarios, particularly the personal garage. Worst-case exposure will probably occur in the personal garage immediately after ignition turn-off when a vehicle produces "hot-soak" evaporative emissions. In most cases, however, personal garage exposures are unlikely to last more than several minutes. The EPA projects that worst-case (i.e., hot-soak) personal garage exposure levels (those from a malfunctioning vehicle in an

unventilated garage) may be as high as approximately 240 milligrams per cubic meter (mg/m^3) of methanol, but that, under more realistic conditions (normal ventilation), levels are unlikely to exceed $130 \text{ mg}/\text{m}^3$. For the traffic situations evaluated, methanol concentrations are projected to be much lower (less than $6 \text{ mg}/\text{m}^3$), even if the fleet is 100% methanol-fueled. One other exposure situation that merits attention is that in which a customer at a self-service filling station will be exposed to roughly $50 \text{ mg}/\text{m}^3$ of methanol vapor for 3 to 4 minutes during refueling. The personal garage and self-service refueling scenarios are important not only because they represent relatively high exposure levels, but also because the methanol concentration, in these cases, is independent of the penetration of methanol-fueled vehicles into the fleet. As a point reference, the American Congress of Governmental Industrial Hygienists' (ACGIH) threshold limit value (TLV) for occupational exposure is $260 \text{ mg}/\text{m}^3$ (200 ppm); this standard is a time-weighted average for an 8-hour period.

Toxicity of Methanol

Nearly all of the available information on methanol toxicity in humans is related to the consequences of acute, rather than chronic, exposures. Acute methanol toxicity evolves in a fairly well-defined pattern. A toxic dose results from intake of a large quantity of methanol in a short period of time, and initially produces a transient, mild depression of the central nervous system. An asymptomatic latent period follows, and may last from several hours to two days or more. The latent period gives way to the onset of a syndrome that consists of an uncompensated metabolic acidosis with superimposed toxicity to the visual system. The physical symptoms, in severe cases, may progress to coma and death; for those who survive, the visual symptoms may, within days to weeks, reverse or progress to permanent visual impairment. The effects that appear after the latent period are attributable to metabolites of methanol, most prominently, formic acid (which dissociates to formate plus a hydrogen ion), and not to methanol itself.

The minimum lethal dose of methanol (in the absence of medical treatment) ranges between 0.3 and 1.0 grams per kilogram of body weight (g/kg). The maximal dose of methanol expected in the EPA's exposure scenarios, by comparison, is under 1 milligram per kilogram of body weight ($0.001 \text{ g}/\text{kg}$). However, the clinical literature indicates that susceptibility to methanol's sub-lethal acute effects may vary widely among individuals. Two of the known determinants of susceptibility are (1) co-exposure to ethanol, which greatly slows methanol's entrance into its metabolic pathway, and (2) the level of liver folate, which is crucial to the oxidation of formate, the key toxic metabolite of methanol.

Until the 1950s, a major obstacle to understanding and treating methanol poisoning was the lack of understanding of some of the mechanisms of methanol toxicity. This situation existed because of the lack of appreciation that non-primate species are not suitable models of acute human methanol toxicity. Gilger and Potts, in 1955, demonstrated that, of all common laboratory species tested, only non-human primates experience methanol toxicity, including ocular pathology, which is characteristic of humans. The non-human primate model has been confirmed, and has enabled a systematic exploration of the metabolic bases, kinetics, and mechanisms of methanol's acute toxic syndrome.

Data on humans or non-human primates exposed to low levels of methanol vapors are scarce and not well-developed. The epidemiologic literature provides weak suggestive evidence that prolonged occupational exposure to methanol vapors at levels above the TLV (260 mg/m³) may produce symptoms such as headache and blurred vision. However, the conclusions are based on symptom reporting, a less preferable source of data than clinical examination, and assessments of exposures are generally inadequate.

In human clinical experiments, two separate Russian studies reported effects of low-level, short-duration methanol exposures (less than 10 mg/m³, approximately 5-minute exposures) on neurobehavioral endpoints, specifically, dark adaptation and EEG-conditioned thresholds. These reports, however, fail to provide descriptions of critical methodological and analytical procedures, as well as complete descriptions of study subjects, and provide only limited data that describes the results. Upon close examination, the results from the two studies are not consistent, and they are not entirely plausible.

In acute and chronic animal experiments published to date in the peer-reviewed scientific literature, there are no indications that adverse health effects are expected at the potential methanol exposure levels discussed earlier. In Japan, the Institute for Applied Energy, with sponsorship of the New Energy Development Organization (NEDO), conducted an extensive research program in which rodents and non-human primates were exposed to methanol vapors either briefly or for extended periods of time. Although the report issued by NEDO indicates potential effects to the central nervous system of non-human primates exposed to 13 mg/m³ for extended periods of time, the details available in that document are insufficient to permit critical evaluation. Further evaluation of these studies will be necessary.

Finally, in people, both methanol and its toxic metabolite, formate, are present at background levels that result from normal dietary intake and natural metabolic processes. A major contributor to the body burden of methanol in many people is the artificial sweetener, aspartame, now found in many foods. Following ingestion, 10% of the aspartame molecule enters the circulation as methanol.

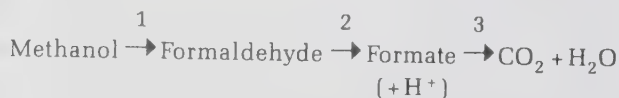
Metabolism of Methanol and Mechanisms of Toxicity

Methanol distributes readily and uniformly to all organs

and tissues in direct relation to their water content. For short-term inhalation exposures, an upper-bound estimate of initial body burden assumes total absorption of the inhaled vapor. A 70 kg person breathing at a ventilation rate of 20 m³/day (twice resting), who is exposed to 200 mg/m³ methanol vapor for 15 minutes (as in a worst-case hot-soak garage scenario), accumulates a methanol body burden of 0.0006 g/kg — at least 500 times lower than doses of acute clinical significance.

Following its uptake and distribution, methanol clears from the human body with half-times of a day or more for high doses (greater than 1 g/kg), and about three hours for the low doses of relevance to this report's objectives (less than 0.1 g/kg). Methanol is either excreted unchanged, mainly in urine and exhaled breath, or enters a metabolic pathway (in the liver) whose ultimate products are carbon dioxide (which is exhaled harmlessly) and water. For the body burdens of methanol that follow a worst-case exposure, metabolism is the dominant pathway, accounting for over 90% of methanol's clearance. This is a key point because methanol's toxic properties are linked to intermediate metabolites, not to the alcohol itself.

In all mammalian species studied, the sequence of metabolic intermediates leading from methanol to its end products is the same:



The toxic properties of methanol, and the basis of species susceptibility, are rooted in the factors that govern the relative rates of formic acid generation (steps 1 and 2) and formate oxidation (step 3). In short, the toxic syndrome sets in if formate generation continues at a rate that exceeds its rate of metabolism to carbon dioxide (CO₂). This imbalance, if protracted, leads to an accumulation of formate coupled, eventually, to an uncompensated metabolic acidosis. The acidosis, if untreated, can prove lethal; formate, even at physiologic pH, is associated with ocular toxicity. In both rats, which are methanol-resistant, and in non-human primates, which are susceptible, the folate pathway in the liver mediates formate metabolism to carbon dioxide. The efficiency of this process is linked to the availability of tetrahydrofolate (THF), the molecule that initially complexes with formate. Non-primate species dispose of formate efficiently at any dose and, thus, escape toxicity, whereas, at sufficiently high doses, humans and non-human primates accumulate toxic metabolites and, thus, are at risk to adverse consequences. As an aside, formaldehyde is not believed to play a role in methanol toxicity.

The mechanisms responsible for injury to the visual system in acute methanol poisonings are not yet understood, but several investigators have postulated that formate, at sufficiently high blood levels, may inhibit cellular respiration in the proximal portion of the optic nerve, leading to a compression type of injury to the nerve's axons that ultimately affects vision. An acidotic state may accelerate such an injury.

Although formate possesses toxic potential, the levels it will achieve in people following worst-case environmental expo-

tures to methanol will not come close to challenging the metabolic capacity of the folate pathway. The small increases of formate levels that have been observed in the blood and urine of adult humans following either occupational exposure to methanol vapors or experimental administration of aspartame reflect normally operating metabolic pathways. The blood levels of formate that follow worst-case (i.e., hot-soak, personal garage) environmental exposure to methanol vapor will, in all likelihood, not be discriminated from the background level of blood formate.

Evaluation and Recommendations

• Discussion

The implementation of methanol as a vehicle fuel is likely to increase the exposure of the general public to methanol vapors. EPA analyses predict that the highest exposure levels will occur in personal garages during engine hot-soak, at the self-service pump during refueling, and, with increasing penetration of methanol technology into the fleet, in public parking garages. By comparison, exposure concentrations in traffic situations, even with 100% penetration of methanol-fueled vehicles, will be very low.

The health effects of methanol are best recognized and studied for cases in which subjects have orally ingested large single doses. The clinical literature documents many case histories of methanol poisoning; its course, which consists of metabolic acidosis and visual disturbance that follow a symptomless latent period, is well characterized. Methanol's toxicity, in these cases, is attributable to its metabolite, formate. Methanol as an unmetabolized substance is not considered toxic unless it is taken in narcotic doses. The discovery, in the 1950s, of the non-human primate as a model of acute human toxicity was perhaps the single most important event to lead to our current understanding of methanol's acute toxicity.

The characteristics of methanol's chronic effects, on the other hand, are not well known. The literature from studies of non-human primates is of little value in evaluating the dose- and time-effect characteristics for protracted exposures of people. The limited evidence from epidemiologic studies and case reports suggests that chronic effects, if they appear, are similar to those described for acute toxicity (e.g., headache, blurred vision), but are less severe. Thus, acute and chronic effects may share common pathways of action. In the small number of instances that report chronic effects attributable to methanol, exposure levels exceed the ACGIH TLV of 260 mg/m³.

In the worst-case exposure scenario (hot-soak, personal garage), the inhaled body burden of methanol (0.0006 g/kg)

will be approximately equivalent to the pre-existing background levels of methanol (0.0005 g/kg) for a brief period of time following exposure. For self-service refueling, the contribution will be roughly 10 times less. The average daily intake of methanol from aspartame in the diet (approximately 0.0003 to 0.0015 g/kg) is on the same order of magnitude as uptake from a single worst-case exposure in the hot-soak garage. Even more importantly, however, worst-case methanol exposures will not lead to blood formate levels that challenge the folate pathway's capacity to oxidize formate. Furthermore, the increase expected in blood formate following worst-case exposure will be negligible in comparison to the background levels of blood formate.

• Conclusion

Based on the foregoing evidence, if methanol produces health effects in normal subjects at or near the exposure levels of concern, these effects would not likely be attributable to the generation of formate. However, the effects of low-level formate accumulation in potentially susceptible subjects has not been examined.

A firm conclusion about the potential health effects from chronic exposures cannot be drawn yet. To date, no human epidemiologic studies have reported effects that could be linked to chronic methanol exposures below the TLV of 260 mg/m³. However, careful investigations of people exposed chronically to levels below the TLV are not available, and would, no doubt, prove very useful if the levels of exposure were rigorously quantitated.

An analysis of the available peer-reviewed literature produces no evidence upon which to base a conclusion that exposure to methanol vapors will result in adverse health effects. This conclusion applies only to exposures that will occur as a result of methanol's normal use as a vehicular fuel, and not to exposure that may occur either from ingesting methanol fuels or from spillage.

Although adverse effects have not been indicated in this analysis, further research targeted to answer specific questions would help in further reducing the uncertainties of estimating the health effects of protracted or repeated low-level exposures, and would serve to reinforce the certainty of conclusions about the public's health. Such research should attempt to elucidate the potential consequences of protracted or repeated low-level exposure, using human epidemiologic approaches and animal experimentation. In the latter, further work could be conducted that would more completely describe the dose- and time-effect relationships between formate concentrations in the body and effects to the visual system. Achieving these objectives would lead to a better understanding of metabolic processes in suspected target tissue.

AUTOMOTIVE METHANOL VAPORS AND HUMAN HEALTH:

An Evaluation of Existing Scientific Information And Issues for Future Research

I. INTRODUCTION

OBJECTIVES OF THE REPORT

Intense interest surrounds the potential use of methanol as a principal automotive fuel for the United States in the next century (Anderson, 1984; Gray and Alson, 1985; ARB, 1986). Several experimental fleets of methanol-fueled vehicles are already in operation, and others are planned (ARB, 1986; RD, 1986; CEN, 1986). The Clean Air Act requires that it be established that implementing such a technology on a mass basis will not degrade environmental quality or compromise the public health. Although substituting methanol for currently used fuels is expected to reduce ambient concentrations of criteria pollutants such as particulate matter, oxides of nitrogen, and ozone (Gold, 1985; ARB, 1986), its use may increase airborne concentrations of two gaseous pollutants of potential concern: formaldehyde and methanol (Harvey et al, 1984). Methanol emissions arise from their release as uncombusted material in the exhaust and also from their evaporation during refueling, and following ignition turn-off; evaporative emissions also occur during the daily heating of the fuel tank. Formaldehyde is a by-product resulting from the incomplete combustion of methanol.

This report focuses on the health issues associated with exposure of the public to methanol vapors emitted from methanol-fueled vehicles. Its objectives are (1) to review the nature and mechanisms of methanol toxicity, (2) to evaluate whether or not methanol's known effects might be expected at anticipated levels of exposure, and (3) to identify both the areas in which critical knowledge is lacking, and the research that could supply the needed information. Although issues such as spillage, oral ingestion of methanol's liquid form, and occupational exposures to methanol vapors are not targeted specifically, the analyses presented may, nonetheless, be useful to these situations as well. [The Health Effects Institute is also keenly interested in furthering knowledge of the potential effects of formaldehyde in the context of exposure to emissions from both conventional and methanol-fueled vehicles. In pursuit of that end, HEI issued Requests for Applications (RFA) to the scientific community in 1985 to study formaldehyde's health effects. The research based on that RFA recently has recently been initiated.]

ORGANIZATION OF THE REPORT

A comprehensive assessment of the potential health impacts of exposure to methanol vapor and a statement of recommendations for further research require integration of information from a variety of sources. Therefore, the next three sections of this report present topics and issues that constitute the building blocks of an environmental health risk assess-

ment of methanol. The final section organizes and synthesizes the technical material in an analytical framework, and provides conclusions and recommendations. A brief description of each section follows:

Section II - Methanol's Fuel Properties and Anticipated Ambient Levels: This section reviews the characteristics of methanol that recommend it as a vehicle fuel, and summarizes studies conducted by the EPA that project the ambient concentrations of methanol that will result from its use. These projections cover various exposure scenarios (e.g., expressway, personal garage) and are critical to help assess whether or not people are likely to experience health effects under anticipated exposure conditions.

Section III - Toxicity of Methanol: This section discusses the signs and symptoms of methanol toxicity, a subject of numerous publications in the toxicologic and clinical literature since the turn of the century. This literature is almost exclusively concerned with the acute effects of methanol poisoning following brief intakes of large quantities of methanol, and supplies very little information about chronic, low-level exposure. The section highlights the differences between primate and non-primate species in terms of their sensitivity to acute methanol toxicity. Such distinctions are important for subsequent discussions of the metabolism of methanol and its toxic mechanisms in humans. Appendices are provided (a) to review the literature that deals with long-term or repeated human exposures to methanol, (b) to describe in detail two Russian human clinical studies, and (c) to discuss the limited literature on chronic exposures of animals to methanol.

Section IV - Metabolism of Methanol and Its Toxic Mechanisms: The uptake, distribution, and subsequent fate of methanol and its metabolites are described. The discussion includes the biochemical basis for the interspecies differences in susceptibility presented in Section III. This material provides the underpinnings for dose-response relationships whose understanding is vital to the overall purposes of this report.

Section V - Evaluation and Recommendations: Here, the information from the preceding four sections is synthesized to project the extent to which known toxic processes may occur under predicted ambient exposure conditions. The analysis considers inhalation as the principal route of exposure, as well as the metabolic and excretory pathways described in Section IV. Finally, the section identifies areas in which the health database might be improved, and advances recommendations for research opportunities that can help in further reducing uncertainties in estimating the health effects of protracted or repeated low-level exposures to methanol.

II. METHANOL'S FUEL PROPERTIES AND ANTICIPATED AMBIENT CONCENTRATIONS

METHANOL AS A FUEL

Methanol (CH_3OH), also called methyl alcohol, is the simplest of all the alcohols. In its pure form, it is a clear, colorless liquid at room temperature, and has the physical and chemical characteristics listed in Table 1. Additional characteristics of methanol relate to its use as a motor vehicle fuel. Pure methanol has an octane rating of between 106 and 115, and an energy content of about 16 kJoule/cm³; in-use gasoline fuels have octane ratings ranging from 87 to 94, and an energy content roughly twice that of methanol (Reed and Lerner, 1973; ARB, 1986).

Table 1. Physical and Chemical Properties of Methanols

Physical state	Colorless, volatile liquid
Molecular formula	CH_3OH
Molecular weight	32.04
Boiling point	64.7°C
Melting point	-97.8°C
Specific gravity	0.7915 (@ 4°C)
Vapor pressure	160 mm Hg (@30°C)
Refractive index	1.3292 (@ 20°C)
Flammability	
Flash point	12°C
Explosive limits	6.0 to 36.5% volume in air
Ignition temperature	470°C
Percent in saturated air	21.05 (@ 30°C)
Density of saturated air (air = 1)	1.02
Solubility	Miscible with water, alcohols, ketones, esters, halogenated hydrocarbons, and benzene
1 mg/ml < >	764 ppm at 25°C, 760 mm Hg
1ppm < >	1.31 mg/m ³ at 25°C, 760 mm Hg

Source: Rowe and McCollister, 1982

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From its discovery in the early 19th century to the mid-1920s, methanol was made exclusively from the destructive distillation of wood, which led to its well-known name, wood alcohol, a term that persists to the present. Methanol had a variety of personal, commercial, and industrial uses as a solvent, medicinal agent, and as a source of energy. In the last century, for example, methanol was used in France for lighting, heating, and cooking. Facing gasoline shortages in the 1930s, several European countries equipped civilian and military vehicles with wood-burning units that provided power in the form of alcohol vapors (Posner, 1975). For the past six decades, however, most methanol has been made by reacting carbon monoxide (CO) with hydrogen (H_2) at elevated

temperature and pressure, and methanol continues to be used in a wide variety of commercial and industrial applications (e.g., manufacture of formaldehyde, anti-freeze ingredient, denaturant) (Reed and Lerner, 1973; Posner, 1975).

The economic, policy, and social factors connected with methanol's future in the automotive fuel marketplace fall outside of the scope of this report. Suffice to say that methanol combines several important attributes that highlight its potential for mass use, which, in turn, justifies an evaluation of its possible impacts on the public health. Briefly, these attributes pertain to: *Availability* - Methanol's precursors (CO and H_2) are available from a variety of carbonaceous sources, particularly coal, which is in abundant reserve in the United States; *Distribution* - The systems already in place for storage and transport of petroleum products also are able to handle alcohol fuels; *Fleet Adaptability* - Currently used gasoline and diesel engines, with varying amounts of modification, can be converted into efficient methanol-burning engines; also, in-use gasoline engines are adaptable to gasoline-methanol blends; *Air Quality* - Emissions from methanol-fueled vehicles are expected to result in ambient concentrations of criteria pollutants no greater than, and very likely, lower than those that result from gasoline or diesel emissions.

EXPOSURE TO METHANOL VAPOR FROM VEHICLES

Despite projected improvements in air quality relative to regulated pollutants, the introduction of methanol technology may result in an increased exposure of the public to methanol and formaldehyde (Harvey et al, 1984), both of which are currently unregulated as mobile source emissions. The Environmental Protection Agency has correspondingly expressed concern for health effects that may be associated with exposure to each of these substances (Carey, 1983; Harvey, 1983). As mentioned earlier, the Health Effects Institute shares this concern, and already has initiated research on the health effects of formaldehyde. This report, however, evaluates the health consequences of exposure to methanol only.

The critical value of exposure assessments to the characterization of environmental impacts on public health is fully recognized (NRC, 1983). Fortunately, a number of studies conducted by the EPA already have provided data on the ambient concentrations of methanol vapor expected under a wide range of vehicle and traffic conditions (Harvey et al, 1984; Gold, 1985). These data, though not yielding detailed time and activity exposure analyses, have identified situations in which maximal, and perhaps toxicologically relevant, exposures are likely to occur.

The basic approach of these studies is to characterize light- and heavy-duty fleet emissions, and, with air quality modeling programs, use these data to compute expected ambient concentrations of methanol. In the initial steps, exhaust and evaporative emissions from individual vehicles are characterized under various conditions related to both driving cycle and the maintenance level of the vehicle. These data may be derived directly from laboratory testing and measurement of

emissions from sample vehicles. Alternatively, emission values may be based on design targets or pre-set certification standards with offset (i.e., correction) factors applied to project actual in-use performance. Offset factors may reflect expected changes in emissions associated with tampering with emission control devices, or malfunctions resulting from either lack of maintenance or from random causes. (See Appendix I for a further description of offsets.)

The next objective is to translate vehicle-specific emissions into ambient concentrations of methanol vapor. For one important scenario, the personal garage, the emission characteristics of solitary vehicles (not fleets) and garage size and ventilation rates are the key determinants of methanol concentrations.

For traffic and parking garage situations, characterizations of entire fleets are essential to project air quality data. The EPA has developed MOBILE3, a database program that helps meet this need. MOBILE3 continually updates detailed profiles of the composition and emissions of in-use gasoline and diesel fleets, and allows for projecting fleet characteristics into the future. The data in the program include miles traveled per vehicle type and model year. For the calculation of total fleet emissions, MOBILE3 factors in variables, such as vehicle deterioration, that may affect the final emissions inventories. The data generated on methanol vapor concentrations, described below and in Tables 3 through 5, are extracted from the air quality models developed for gasoline and diesel fleets. (Appendix I further describes the exposure scenarios and the data that appear in Tables 3 through 5.)

The EPA studies have modeled methanol exposure levels that may occur in specific situations representative of the full range of vehicle use and traffic flow. Table 2 lists the exposure scenarios for light-duty vehicles together with the driving cycle and type of emissions (exhaust and/or evaporative) that best characterizes vehicle performance within each scenario. The 'typical' and 'severe' classifications for street canyon, roadway tunnel, and expressway in the table refer to traffic density and road design, as well as to the driving speed and test cycle listed. For example, the typical street canyon scenario that is modeled calls for a sidewalk exposure next to a four-lane street with a traffic load of 800 vehicles per hour; in the severe case, the street is six lanes with 2,400 vehicles per hour.

Although not shown in Table 2, typical and severe emissions also apply to the personal and parking garage scenarios. For personal garages, vehicle warm-up time influences the extent of potential exposure during the idle cycle; for example, in the model, a typical warm-up interval for moderate weather conditions is 30 seconds, and is 5 minutes during severe winter conditions (for both, the garage door is considered to be open). During personal garage hot-soak (evaporation from a hot engine after it has been turned off), the relative severity of exposure is a function of a garage's size and air exchange rate. For parking garages, inflow and outflow rates of traffic, as well as facility size and ventilation, all determine idle and hot-soak concentrations.

Table 2. Scenarios and Appropriate Test Cycles for Evaluating Emissions from the Use of Methanol as a Vehicle Fuel

Scenario	Scenario Average Speed	Suggested Test Cycle	Test Average Speed
Street Canyon			
Typical	5	NYCC ^a (+ SHED) ^b	7.1
Severe	20	FTP ^c (+ SHED)	19.5
Tunnel			
Typical	0-40	FTP (hot) ^d	19.5
Severe	25	FTP (hot)	19.5
Expressway			
Typical	55	HFET ^e	48.2
Severe	50	HFET	48.2
Off Road			
Parking and Personal Garages (idle)	0	Cold Idle	0
Parking and Personal Garages (hot-soak)	0	Hot-Soak (SHED)	0

^a NYCC: New York City Cycle, a low speed driving cycle (exhaust emissions)

^b SHED: Sealed Housing for Evaporative Determination, the standard evaporative emissions test

^c FTP: Federal Test Procedure, a cold start driving cycle simulating typical urban driving (exhaust emissions)

^d FTP(hot) refers to a hot start FTP (i.e., vehicle already fully warmed up) (exhaust and evaporative emissions)

^e HFET: Highway Fuel Economy Test, a driving cycle simulating rural highway driving (exhaust emissions)

Source: Harvey et al, 1984

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Tables 3 through 5 display the methanol concentrations expected under the conditions described above. Although the discussion above focused on light-duty vehicles, the values in Table 3 (roadway traffic scenarios) represent combined light- and heavy-duty fleets; Tables 4 and 5, which describe personal and parking garage scenarios, represent exposures from only light-duty vehicles. The tables show that maximal exposures are expected in the garage scenarios, particularly in the personal garage. In a public parking facility, exposure levels may reach as high as 60 mg/m³ during hot-soak, assuming all of the vehicles in the garage are fueled with methanol. In a normally ventilated personal garage, methanol concentrations produced by hot-soak emissions from a malfunctioning vehicle (i.e., one with a disabled canister) probably will not exceed 150 mg/m³. For the hypothetical case of hot-soak emissions from a malfunctioning vehicle in an unventilated garage, the methanol concentration may reach as high as 240 mg/m³, a level that may be considered at the outer limits of exposure. In most cases, however, personal garage exposures will be brief, lasting only for the period that the operator occupies the garage during idle or hot-soak, perhaps

Table 3. Estimated In-use Ambient Methanol Concentrations for Traffic Scenarios — mg/m³
(100% Fleet Penetration)

Scenario	Likely Certification Level (g/mile)	All Vehicles Meet Standard ¹	25% of Vehicles Malfunction ²
STREET CANYON			
Typical	0.54	0.04	0.08
Severe		0.25	0.54
ROADWAY TUNNEL			
Typical	0.54	1.0	2.1
Severe		2.5	5.4
EXPRESSWAY			
Typical	0.023	0.005	0.19
Severe		0.019	0.77
Off-road		0.004	0.161

¹ - fleetwide offset = 1.0

² - fleetwide offset = 2.2, street canyon
2.2, roadway tunnel
40.8, expressway

Data adapted from: Gold, 1985 and Harvey et al, 1984

NOTE: For a further explanation of the data, a description of the scenarios, and assignment of offset factors, see Appendix I.

Table 4. Estimated Peak In-use Ambient Methanol Concentrations for Personal Garage — mg/m³
(Solitary Vehicle)

Mode	Likely Certification Level	Vehicle Meets Standard ¹	Vehicle Malfunctions ²
IDLE (TRIP START)			
Typical	0.002-0.043 g/min	0.02-0.34	0.54-12
Severe		0.13-2.9	4.6-98
HOT-SOAK (TRIP END)			
Typical	1.1-3.0 g/hr	10-27	48-130
Severe		18-49	86-240

¹ - vehicle offset = 1.0

² - idle offset = 34 (non catalyst configuration)
hot-soak offset = 4.8 (canister disabled)

Data adapted from: Gold, 1985 and Harvey et al, 1984

NOTE: Exposure in the personal garage scenario is brief, probably not lasting more than 15 minutes. For a further explanation of the data, a description of the scenarios, and assignment of offset factors, see Appendix I.

a matter of minutes. For traffic situations (Table 3), methanol concentrations are generally much lower (less than 6 mg/m³) than in the garages, with maxima predicted for roadway tunnels.

Table 5. Estimated In-use Ambient Methanol Concentrations for Parking Garage — mg/m³
(100% Fleet Penetration)

Mode	Likely Certification Level	All Vehicles Meet Standard ¹	Vehicle Malfunction ²
IDLE (TRIP START) ³			
Typical	0.002-0.043 g/min	0.17	1.6
Severe		2.4	23
HOT-SOAK (TRIP END)			
Typical	1.1-3.0 g/hr	0.30-0.81	0.49-1.4
Severe		13-35	21-58

¹ - fleetwide vehicle offset = 1.0

² - fleetwide offset for idle = 9.5 (based on 25% malfunction rate)
fleetwide offset for hot-soak = 1.7 (based on 10% malfunction rate)

³ - Idle data based on upper certification level

Data adapted from: Gold, 1985 and Harvey et al, 1984

NOTE: Exposure in the parking garage scenario is brief, probably not lasting more than 15 minutes. For a further explanation of the data, a description of the scenarios, and assignment of offset factors, see Appendix I.

All scenarios except the personal garage assume 100% penetration of methanol-fueled vehicles into the fleet. Thus, all methanol values in Tables 3 and 5 scale linearly with penetration fraction. Despite the brief exposure interval, the personal garage scenario is important not only because it represents the highest exposure level, but also because the methanol concentration is independent of penetration. Although the fleet values (Tables 3 and 5) may not be achieved for decades, if ever, those for personal garages (Table 4) are projected for the individuals driving methanol-fueled cars and using personal garages at any level of penetration. Therefore, the potential exposures in personal garages may be of more immediate concern.

One final exposure situation that merits attention is service-station refueling. According to EPA estimates, a typical methanol fill-up will result in about three to four minutes of exposure to between 33 and 50 mg/m³ of methanol (Harvey et al, 1984). For self-service customers, such exposures may occur once or twice weekly, but for station attendants exposure will be much more frequent.

The exposure data described above in Tables 3 through 5, and in Appendix I, are summarized in Figure 1. For reference, Figure 1 also indicates that the American Congress of Governmental Industrial Hygienists' (ACGIH) threshold limit value (TLV) for exposure to methanol averaged over an 8-hour work day is 260 mg/m³ (ACGIH, 1985). This value has been designated for working populations, and not for the general public, for whom the philosophy of standard-setting differs. It is presented only as a point of reference for the ambient exposure data in Figure 1.

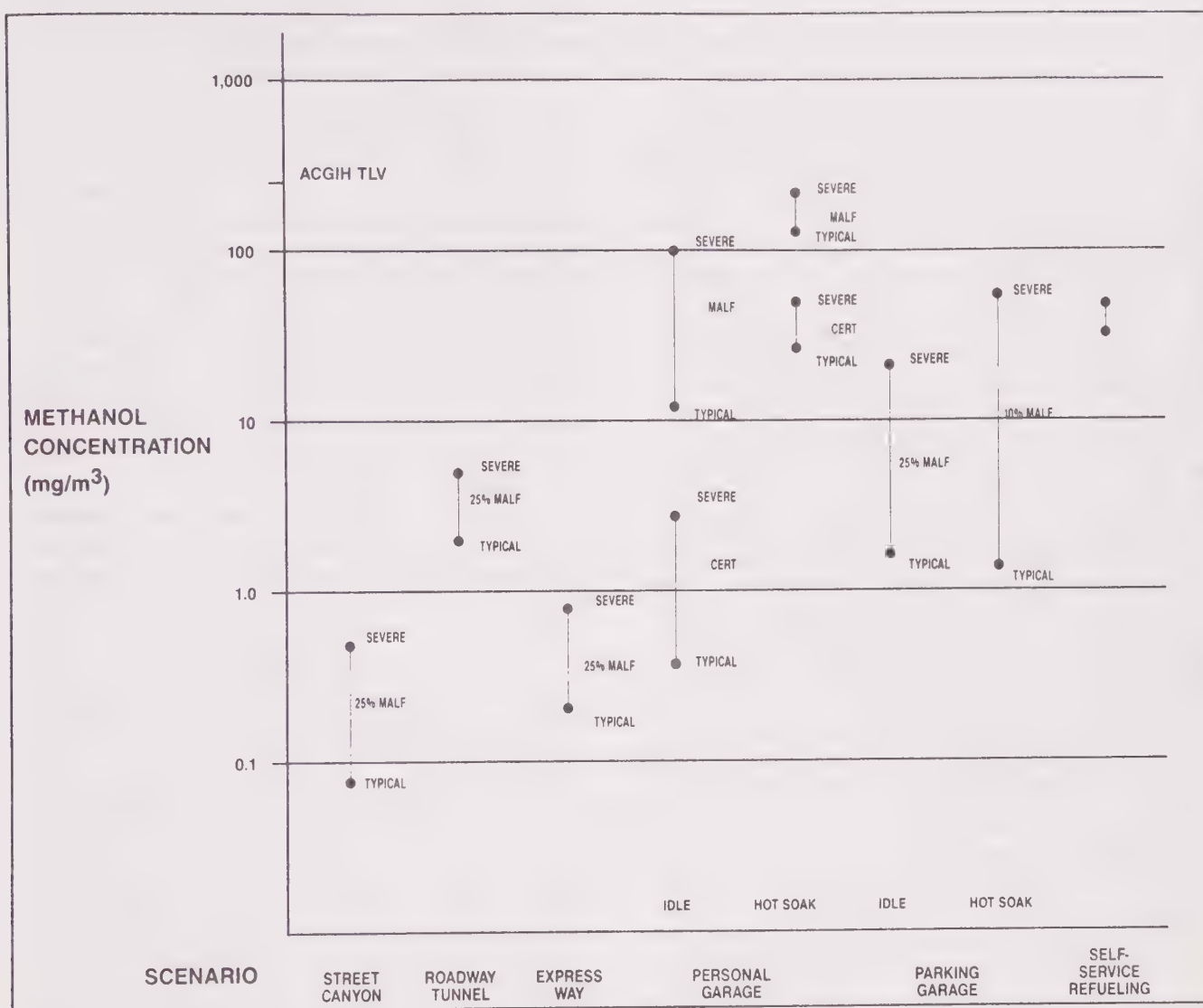


Figure 1: A summary of the data on methanol exposure levels shown in Tables 3 through 5. Severe (S) and Typical (T) relate to conditions described in Section II and Appendix I. For street canyon, roadway tunnel, expressway and parking garage (idle), the data are based on a 25% malfunction (Malf) rate in the fleet. For parking garage (hot soak), the data are based on a 10% malfunction rate. Because a personal garage scenario involves only a single vehicle, data are shown for a malfunctioning vehicle and for one operating according to likely certification standards (Cert). In this case, the above designations do not apply to the refueling scenario.

III. TOXICITY OF METHANOL

BACKGROUND

In the 1890s, the use of wood alcohol increased significantly, and its acute toxicity was fully realized shortly thereafter. Previously, impurities retained in its distillation process rendered wood alcohol a vile-tasting and foul-smelling substance. Human exposures to wood alcohol and its vapors consequently were limited, and reports of intoxication, therefore, were very rare. The introduction of an inexpensive deodorization process during the 1890s increased greatly the market for wood alcohol as a commercial product and as a solvent for use in the workplace. It was sold in stores as a pure substance under various commercial names such as Columbian Spirits, Eagle Spirits, and Lion d'Or, and was included as an

ingredient in many other consumer products such as witch hazel, Jamaica ginger, vanilla extract, and perfumes (Jelliffe, 1905; Wood, 1906). Perhaps the most notorious use of wood alcohol was, and continues to be, as an adulterant in alcoholic beverages, a practice that has led to large scale episodes of poisonings since the turn of the century (Chew et al, 1946; Province et al, 1946; Bennett et al, 1953; Kane et al, 1968; Dethlefs and Naraqi, 1978).

In parallel with its spread in the consumer market place, wood alcohol became a widely used substance in the workplace. Tyson and Schoenberg (1914) reviewed data from the 1904 U.S. census, and tallied nearly two million workers in occupations in which wood alcohol was used. Those most heavily represented included: painters, glaziers, and varnishers (278,000); launderers (386,000); boot and shoemakers (200,000); and printers and lithographers (155,000).

The dramatic increase in wood alcohol's distribution and use, coupled with an almost universal ignorance concerning its toxic potential, led quickly to an accumulation of case reports describing wood alcohol poisonings. In 1904, Wood and Buller published an oft-quoted series of 235 case studies that characterize many of the key presenting features of acute methanol poisoning (Wood and Buller, 1904; Buller and Wood, 1904). Briefly, about a day after exposure, victims are stricken with visual disturbances and an array of incapacitating physical symptoms that may lead to coma and death. Wood and Buller and others in the medical community sounded alerts concerning wood alcohol and lobbied in the medical literature for social and legislative actions to control access to wood alcohol and its use (Wood, 1912; Tyson and Schoenberg, 1914; Ziegler, 1921). The vast majority of poisonings in Wood and Buller's reports, and in the many wood alcohol/methanol incidents recorded since, have occurred from drinking adulterated beverages or wood alcohol products. In the largest single episode, Bennett et al (1953) describe a case that occurred in Atlanta in 1951 when, within a five day period, 323 people ingested bootleg whisky contaminated with methanol; 41 of these poisonings were fatal.

Although oral ingestion dominates historically as the most frequent route of poisoning, the literature also substantiates that percutaneous absorption of methanol liquids or inhalation of its vapors are as effective as the oral route in producing methanol's acute toxic syndrome (Wood and Buller, 1904; Buller and Wood, 1904; Gimenez et al, 1968). Tyson and Schoenberg (1914) counted about 100 cases reported up to 1912 of amblyopia (impairment of vision) and death from inhalation of wood alcohol vapors. Referring to the case literature on wood alcohol inhalation Ziegler (1921) wrote:

"The majority of these cases occur from occupational exposure to the fumes. The painter uses it as a cleansing fluid or as a cheap diluent to cut his shellac in order to varnish the interior of large beer vats, closets or closed room. Two of Tyson's (1912) patients finally succumbed to the slow poisoning. The latter mixes it with shellac to stiffen the nap or straw blanks. The dyer of feathers uses it to dilute the colors; the maker of shoe polish adds it to the paste; the brass finisher uses it in the lacquer, and the maker of rubber tires mixes the mass with it. If ventilation is very free, the danger will be lessened; but open air exercise at frequent intervals should be required for every such employee."

ACUTE TOXICITY: DESCRIPTION

Nearly all of the available information about methanol toxicity in humans concerns the consequences of acute exposures.¹ This information is based on clinical case studies recorded since the turn of the century and, more recently, on laboratory experiments that employ valid animal models of human toxicity. The few reports in the human case literature concerning repeated or prolonged exposures suggest that

chronic and acute effects may share similar qualities. Therefore, a description of methanol's acute toxic properties is appropriate to this report's objectives.

Acute methanol toxicity in humans evolves in a fairly well-defined pattern. A toxic exposure results initially in a transient, mild depression of the central nervous system (CNS). An asymptomatic latent period follows, and may last from several hours to two days or more, although 12 to 24 hours is most common (Bennett, 1953; Roe, 1955). The latent period gives way to the onset of a syndrome that consists of an uncompensated metabolic acidosis with superimposed toxicity to the visual system. Physical symptoms typically may include headache, dizziness, nausea, and vomiting; these may be followed by severe abdominal pain and difficult, periodic breathing (Kussmaul breathing), which may progress to coma and death, usually from respiratory failure (Tephly and McMartin, 1984; Jacobsen and McMartin, 1986).

In parallel with the onset of these symptoms, subjects experience visual disturbances that include blurred or indistinct vision and altered visual fields (often depression of the central field), and, in severe cases, total blindness (Chew et al, 1946; Bennett et al, 1953). Impairment of the pupillary response to light usually accompanies the visual symptoms, and the extent of impairment is predictive of survival. Subjects with unresponsive, dilated pupils often succumb to the toxic syndrome, and those who survive suffer appreciable and, in many cases, permanent loss of vision.

Ophthalmoscopic examinations of methanol-poisoned victims show that hyperemia (i.e., a local increase in blood flow) of the optic disc is the earliest change that occurs in the retina; hyperemia accompanies the initial visual symptoms (Benton and Calhoun, 1952; Dethlefs and Naraqi, 1978). Within a day, a white striated edema (an accumulation of an excessive amount of watery fluid) appears that projects into the surrounding retina from the optic disc, whose margin simultaneously acquires a blurred appearance; the papilla itself is not edematous. (The papilla, also called the nerve head, is the area where the nerve fibers of the retina converge to form the optic nerve.) The optic disc hyperemia usually subsides within a week, but edema in the region of the optic disc may persist for up to two months. The edema follows the course of major blood vessels and appears to be located mainly in the nerve fiber layer of the retina.

In the Atlanta epidemic, these ophthalmoscopically visible changes were observed in 87% of patients with acute visual symptoms, and in all patients who developed permanent visual deficit. Furthermore, the severity of retinal edema was predictive of restoration of vision; mild edema resulted frequently in full recovery, and severe edema led invariably to permanent effects (Benton and Calhoun, 1952). Pallor of the optic disc is an end-stage sign of irreversible effects to the visual system, and may appear one to two months after an acute methanol dosage (Wood and Buller, 1904; Buller and Wood, 1904; Bennett et al, 1953) [or possibly following chronic

¹In this report, the terms acute and chronic refer to the time-course of exposure, rather than to the time-course of the appearance of effects. The reader should bear this distinction in mind as methanol's best-characterized effects appear after a latent period of about a day following a single (i.e., acute), large exposure, and may continue to develop or persist for days to weeks or longer.

occupational exposure to methanol vapors (Tyson, 1912; Ziegler, 1921)]. The pallor indicates a loss of the blood supply to the head of the optic nerve, and frequently reflects atrophy of the optic nerve (Casarett and Doull, 1975).

Autopsies from victims of lethal methanol poisonings have revealed gross pathology in the visceral organs, the lung, and the CNS, all of which involve a variety of edematous, hemorrhagic, and degenerative changes (Tonning, 1945; Chew et al, 1946; Province et al, 1946; Bennett et al, 1953; Kaplan, 1962; Erlanson et al, 1965). Several case studies report post-mortem signs of damage to the basal ganglia in the brain, specifically the putamen (Province et al, 1946; Erlanson et al, 1965; Aquilonius et al, 1978). This area of the brain participates in the control of gross intentional motor activities that are normally performed unconsciously. Damage to this area results in various motor disorders such as Parkinsonism and Huntington disease. A number of human studies have shown that survivors of severe methanol intoxication may suffer residual damage to the putamen and have associated motor disorders (Erlanson et al, 1965; Guggenheim et al, 1971; Aquilonius et al, 1978; Ley and Gali, 1983).

NATURE OF ACUTE TOXICITY

At this point, it is appropriate to summarize the metabolic basis of methanol toxicity, discussed in detail in the next section. Briefly, the metabolites of methanol, not methanol itself, are responsible for the toxic syndrome of acidosis and ocular toxicity that appear after the latent period (Kane et al, 1968; McMartin et al, 1980; Sejersted et al, 1983; Osterloh et al, 1986). Following uptake, methanol is metabolized in the liver to carbon dioxide (and water) through several enzymatic steps, and the carbon dioxide then is exhaled harmlessly (Tephly and McMartin, 1984). Formic acid is an intermediate product in the metabolic sequence, and, at physiologic pH, dissociates quickly and almost completely to its constituent formate and hydrogen ions. These ions then may accumulate in the body if the throughput of methanol exceeds the rate that formate is oxidized to carbon dioxide. Two related events then occur: (1) an acid load is imposed systemically, which, if persistent, eventually overwhelms acid-base homeostasis — this disorder requires roughly a day to develop and accounts for the latent period; and (2) formate exercises a localized toxic effect in the anterior region of the optic nerve by mechanisms that are still unclear.

Two facets of methanol toxicity that were appreciated quite early concern, first, the dose level of methanol that is hazardous to humans, and, second, the variable susceptibility to acute effects among individual people. Reflecting on his collection of case studies published with Buller, Wood stated in 1912:

“As in the case of several other poisons, some persons are largely immune so far as permanent damage to the organism is concerned. If ten persons drink, say, four ounces of Columbian spirits within three hours, all will have marked abdominal distress and four will die, two

of them becoming blind before death. Six will eventually recover, of whom two will be permanently blind before death. With still larger doses, the proportion of death and blindness will be greater.”

In this summary statement, 4 ounces of Columbian spirits, or 95 grams of methanol (Columbian spirits is basically pure methanol) is lethal to 40% of the cases; for a 70 kg person, this dose is equivalent to about 1.4 grams of methanol per kg of body weight (g/kg). This figure is consistent with currently accepted values for lethality, and 0.3 to 1 g/kg is considered the range of a minimum lethal dose for untreated cases (Roe, 1955; Erlanson et al, 1965; Koivusalo, 1970; Gonda, et al, 1978).

As mentioned, the time to onset of symptoms among poisoned victims is quite variable, ranging from several hours to a few days. The variability of the effective dose is a prominent feature of acute methanol toxicity as well (Wood and Buller, 1904; Buller and Wood, 1904; Chew et al, 1946; Tephly and McMartin, 1984). A case report of poisoning among American soldiers in 1945 states that for each patient admitted to the hospital, up to four others had taken equivalent amounts of methanol without seeking medical care (Province et al, 1946). In the Atlanta epidemic, most patients claimed drinking about a quarter of a pint (approximately 125 cc) of “moonshine,” which consisted of 40% methanol. The lowest lethal dose reported was “three teaspoons (about 15 ml),” and the largest dose survived was about a half-liter of this mixture.

Variable susceptibility is a hallmark of human and animal responses to virtually all toxic substances. The elucidation of the root causes of variability is essential to evaluate the potential public health impacts of substances likely to be ubiquitous, such as community air pollutants. Two general classes of factors determine susceptibility: metabolic and physiologic characteristics that are inherent (e.g., age, sex, genetic determinants), and the effects of external influences such as lifestyle and exposure to other substances. In the case of methanol toxicity, susceptibility factors of both classes no doubt remain unidentified. Two that are known to influence sensitivity are the amount of ethanol ingested with methanol, and the dietary sufficiency of folate.

How do ethanol and folate contribute to inter-individual variability? In humans, the hepatic enzyme that first processes methanol, alcohol dehydrogenase (ADH), also oxidizes ethanol. However, ethanol's affinity for ADH is 5 to 10 times greater than methanol's (Kini and Cooper, 1961; Makar et al, 1968). As long as ethanol persists in the circulation, it retards methanol's entrance into its metabolic pathway, thereby slowing the formation of formic acid. Thus, patients who simultaneously drink toxic doses of methanol with large amounts of ethanol, as has frequently occurred, may be spared methanol toxicity, or alternatively, may experience a delayed onset of its symptoms (Roe, 1955).

In the liver, formate is metabolized to carbon dioxide via a folate-dependent pathway. Research with animals has demonstrated that folate deficiency predisposes to the accumulation of formate, and, therefore, to a state of heightened

susceptibility to methanol toxicity (McMartin et al, 1977a; Makar and Tephly, 1977). In all likelihood, humans possess similar characteristics.

To date, modulators of susceptibility are described only for acute effects of methanol. Though these factors also may play roles in differential susceptibility to chronic effects, the list of factors that modify responses to methanol cannot be considered complete.

DEVELOPMENT OF ANIMAL MODELS

Extensive clinical and research efforts in this and other countries have been invested to understand the toxicologic, biochemical, and metabolic nature of methanol intoxication, and to improve the management of methanol-poisoned patients. In the early part of this century, the prognosis for methanol intoxication was poor, and available treatments were ineffectual. Since the 1940s, clinical advances have quickened, and the loss of vision and life from methanol exposure has abated significantly. Today, three therapeutic modes generally are used, usually in concert to alleviate the signs and symptoms of acute methanol toxicity. They are: (1) alkali treatment to restore acid-base balance, (2) ethanol treatment to retard the metabolism of methanol, and (3) hemodialysis to accelerate the clearance of methanol and formate from body fluids. (Peritoneal dialysis, less effective than hemodialysis, is also used on occasion.) Other potential treatments are still in the research or trial stages (Jacobsen and McMartin, 1986).

For the half century that followed Wood and Buller's 1904 reports, a major impediment to understand and treat methanol poisoning was the lack of awareness that only non-human primate species presented a valid model of acute human methanol toxicity. In their report on ocular effects in the Atlanta epidemic, Benton and Calhoun (1952) wrote: "It is unfortunate that laboratory animals do not respond to this agent in a manner similar to the human. Acidosis almost never develops, and the animals do not often appear to go blind."

In 1955, Roe, who in the 1940s first advocated ethanol therapy, wrote: "It is a waste of time to attempt to investigate the mechanism of the toxic effects of methanol in man by means of animal experiments until it is clear why animals do not develop more than a moderate degree of acidosis." The term "animal" in both of these quotes refers to non-primate species. Most animal research to that time focused on dogs, rabbits, rats, and mice; the few results from experiments on non-human primates did not emerge as unique. In 1955, Gilger and Potts published a landmark paper that, for the first time, established the non-human primate as the model of choice for acute methanol toxicity in humans. The paper's introduction provides an excellent review and critique of the extant literature on methanol toxicity in animals, and indicates this literature's technical inadequacies and the misconceptions it helped to create. In the experiment, Gilger and Potts (1955) exposed rats (Sprague-Dawley), rabbits, dogs, and rhesus macaque monkeys to a range of methanol doses. (They also exposed mice as reported in a previous paper (Gilger et al,

1952). Gilger and Potts observed that (1) the lethal dose for non-primates was two to three times higher than the 3 g/kg lethal dose reported for the monkeys (i.e., 6 to 10 times higher than lethal doses in humans), and (2) of all the species tested, only non-human primates experienced a sequence of early inebriation, then a one day latency followed by the toxic syndrome characteristic of humans (acidosis, some ocular toxicity), which precede their death; the other species developed an initial narcosis from which they either survived or died, and acidosis was not a prominent feature of this toxicity.

In follow-up papers, these investigators studied in non-human primates methanol-induced pathology and the effects of both bicarbonate (i.e., base) and ethanol therapy on the clinical course following lethal-if-untreated doses of methanol (Potts, 1955; Potts et al, 1955; Gilger et al, 1956; Gilger et al, 1959). (All used rhesus macaques except Gilger et al, 1959, who used both rhesus and grivet monkeys.) Both treatments effectively prevented death, as they do in humans, but bicarbonate therapy did not suppress the appearance of retinal edema. However, no signs of ocular toxicity occurred in ethanol-treated animals. These findings are consistent with the principles that (1) acidosis is the proximal cause of general symptoms and death in human methanol intoxication and (2) blocking the metabolism of methanol prevents simultaneously the acidosis and the generation of metabolites toxic to the visual system.

The legitimacy of the non-human primate model has since been confirmed (Clay et al, 1975; McMartin et al, 1975) and has enabled a systematic exploration of the metabolic bases, kinetics, and mechanisms of methanol's acute toxic syndrome, all of which are covered in greater depth in section IV. Research in lower species, primarily rats, has, nonetheless, been integral to the refinement of the non-human primate model. The contrast between the metabolic machinery of rats and non-human primates has helped pinpoint some important biochemical characteristics that influence the sensitivity or resistance to methanol poisoning. Moreover, these advances in animal models lessened the absolute reliance on human data, which are usually collected in the heat of a medical emergency. Finally, non-primates may remain appropriate models in studies that seek to understand the direct alcoholic effects of methanol.

The sub-sections that follow cover specific topics related to methanol toxicity that are relevant to the concerns of this report. Appendices are provided to amplify the discussion, when appropriate.

REPEATED OR PROLONGED HUMAN EXPOSURES TO METHANOL

As mentioned earlier, the information available suggests that extended human exposures to methanol may cause effects qualitatively similar to those from relatively high levels of acute exposure. This information is based on a limited number of case reports and even fewer epidemiologic studies. This literature, summarized in Appendix II, suffers generally from

the classic shortcomings that include unknown levels and/or durations of exposure. Nevertheless, taken together, the studies of Kingsley and Hirsch (1955), NIOSH (1981), and Frederick et al (1984), suggest that chronic exposures above 200 ppm (260 mg/m³) may produce signs of methanol toxicity, including headache, dizziness, nausea, and blurred vision. The study by Frederick et al (1984) of teacher aides who worked near spirit duplicating machines is, perhaps, the most useful study, and is reviewed in considerable detail in Appendix II. In the only no-effect study that provided ambient measurements, methanol levels were about 30 mg/m³ (Greenburg et al, 1938). For reference, the American Conference of Governmental Industrial Hygienists (ACGIH) TLV, or threshold limit value, is 260 mg/m³ time-weighted average (TWA) over 8 hours (ACGIH, 1985a); also, ACGIH has designated a short-term (15 minute) threshold value of 310 mg/m³. The ACGIH standard is reproduced in Appendix III. In 1976, the National Institute for Occupational Safety and Health (NIOSH) recommended a TWA standard of 260 mg/m³, and a 15-minute ceiling of 1,050 mg/m³ (800 ppm) (NIOSH, 1976a).

RUSSIAN STUDIES OF HUMANS: LOW-LEVEL EXPOSURES

Russian investigators published papers in 1959 and 1967 that claimed neurobehavioral effects in humans exposed to very low exposure levels of methanol vapors (less than 12 mg/m³). These are summarized here and discussed in greater detail in Appendix IV. In the first paper, Chao (1959) measured the threshold of olfactory perception and dark adaptation (or light sensitivity). The second paper by Ubaydullayev (1967), included both of these measures in addition to the EEG conditioned reflex threshold. In the olfactory studies, Chao (13 subjects) and Ubaydullayev (25 subjects) report the range for minimum detectable methanol concentrations of 4.3 to 11.1 mg/m³ and 3.7 to 10.5 mg/m³, respectively, and the range for maximum imperceptible concentrations of 3.7 to 10.5 mg/m³ and 3.9 to 9.7 mg/m³, respectively. Minimum levels of methanol that affected dark adaptation were 3.3 mg/m³ for Chao (3 subjects) and 3.5 mg/m³ for Ubaydullayev (3 subjects). Finally, the latter investigator found the threshold for EEG conditioned reflexes at 1.17 mg/m³ (2 of 6 subjects).

The results of these studies suggest that exposures of several minutes or less to very low concentrations of methanol stimulate visual and peripheral olfactory receptors, and may influence the processing of stimuli in the central nervous system. The two studies, published eight years apart, produced consistent data on olfactory thresholds and qualitatively similar data on dark adaptation.

Appendix IV addresses in greater detail several problems that call the results of these studies into question. Briefly, subject descriptions were not provided, and neither were several important details concerning data acquisition — for example, no information is provided on the specific time points selected for testing dark adaptation, or on the length of exposure in the EEG conditioning test. The purity grade of methanol used

was not described, which leads to a suspicion that impurities in the methanol could have confounded the determination of olfactory thresholds, and possibly the measures in the other two experiments. Also, these investigators did not discuss the manner in which methanol concentrations were measured, another potential source of error. Several features of the two dark adaptation studies display conflicts that cannot be resolved. Finally, the dark adaptation and EEG effects occurred at exposure conditions that would contribute a negligible amount to the background levels of methanol in the body (see below for discussion of background levels).

ANIMAL STUDIES SPONSORED BY NEW ENERGY DEVELOPMENT ORGANIZATION

In Japan, the Institute for Applied Energy, with the sponsorship of the New Energy Development Organization (NEDO) conducted an extensive program in which rodents and non-human primates (cynomolgus monkeys) were exposed to methanol vapors. (In Japan, methanol has been contemplated as a fuel for power plants.) Non-human primates were exposed chronically for up to 30 months (22 hours per day) to 13, 130, and 1,300 mg/m³ of methanol vapors. Other groups of monkeys were exposed for shorter durations (6 days to 7 months) to a higher range of levels (2,300 to 13,000 mg/m³). Also, monkeys were subjected to metabolism evaluations during a 48-hour period that followed intraperitoneal administration of methanol (25 to 3,000 mg/kg).

In the rodent experiments, rats (Fischer 344) and mice (B6C3F1) were exposed to 13, 130, and 1,300 mg/m³ of methanol for 12 months, to examine for toxic effects unrelated to carcinogenesis, and for 24 months (rats) and 18 months (mice) to examine for tumor induction (all exposures were for 20 hours per day). These same exposure levels were used in rat experiments (Sprague-Dawley) that tested for potential effects on reproductive performance over two generations. Teratology experiments also were carried out on Sprague-Dawley rats exposed to 0, 260, 1,300, and 6,500 mg/m³ for days 7 to 17 of gestation. Finally, Fischer 344 rats were subjected to metabolic evaluations like those performed on the macaques.

The NEDO program also included a battery of genotoxic assays, with various cell and bacterial systems exposed to methanol. The summary report issued by NEDO (1986) in general concludes that toxic, reproductive, and carcinogenic effects are not evident at chronic exposure levels of 130 mg/m³, and teratologic effects do not occur at 1,300 mg/m³. However, the report does indicate the possibility of subtle effects in the central nervous system of non-human primates exposed chronically to 13 mg/m³, specifically, the appearance of "reactive astrocytes." These results may or may not be of significance with regard to the exposure of the public to methanol vapors. The authors of the report attach little biological significance to these findings.

Unfortunately, the report does not include a sufficient amount of technical data and histopathological findings to

enable a critical review of the experiments and their results. However, the types of experiments performed are crucial to the evaluation of potential risks from exposure to airborne substances. Perhaps further evaluation of this program's experimental methods and data will help clarify the results observed, and their relationship to methanol exposure.

PROLONGED EXPOSURE TO NON-HUMAN PRIMATES

In a study of relevant concern, newborn stump-tail macaques received aspartame in their formula daily for nine months, starting from between 17 and 42 days of age (Reynolds et al, 1984). Aspartame hydrolyzes in the gut to aspartate, phenylalanine, and methanol. The methanol accounts for 10% of aspartame's molecular weight. The exposed animals ate 1, 2, or 2.5 to 2.7 grams of aspartame/kg/day, which is the equivalent of 100, 200, and 250 to 270 mg methanol/kg/day. The investigators report no effects with respect to growth, hematology, serum chemistry, urinalysis, and EEG patterns. The investigators concluded, "Large intakes of aspartame as part of the diet appear to have no effect upon developmental parameters of the infant macaque." This experiment, however, did not include histopathologic analyses.

These same animals also were subject to hearing tests and a battery of behavioral tests that included object discrimination, pattern discrimination, and learning (Suomi, 1984). Again, the investigators were unable to detect any effects from chronic aspartame ingestion. The test battery, according to the investigators, is one "that previously had been shown to be sensitive to age difference, size and locations of cortical lesions, form of early rearing history, some chronic dietary conditions, and exposure to various environmental toxins."

In a subchronic inhalation study, cynomolgus monkeys were exposed for 4 weeks (6 hours per day, 5 days per week) to 650, 2,600 and 6,500 mg/m³ of methanol vapor (Andrews et al, 1987). The animals were observed twice daily for signs of toxicity, and were given detailed physical examinations each week, and ophthalmoscopic examinations at pre-test and at termination. Following sacrifice, the animals' organs were examined and weighed, and selected tissues from all animals in the control and high exposure groups were examined microscopically. These included nasal turbinates, trachea, lungs, trachea, esophagus, liver, and the eye and optic nerve. No treatment-related effects on cynomolgus monkeys were observed in this study.

PROLONGED EXPOSURE TO NON-PRIMATES

Very few studies have been conducted to investigate the potential effects of long-term exposure of non-primate species to methanol. At this point, there are no firm indications of chronic effects near exposure levels of concern. Details of the pertinent literature are in Appendix V.

FORMIC ACID

Appendix VI briefly reviews the toxicology of formic acid, which is the key toxic metabolite of methanol. The most

important effects of formic acid are, apparently, those linked to its effects on the visual system, discussed in detail in Section IV.

BACKGROUND BODY BURDENS OF METHANOL AND FORMATE

Exposures to substances in the environment often contribute to a pre-existing body burden of that substance or its metabolites, found in people classified as "unexposed." For example, exposures to the airborne pollutants, carbon monoxide and manganese, contribute to background levels that would be found in people breathing clean air.

The two most prominent sources of background body burdens for methanol and formate are diet and natural metabolic processes. Sedivec et al (1981) reported a mean blood methanol level of 0.73 mg/l in 31 unexposed subjects (range: 0.32 to 2.61 mg/l), and Eriksen and Kulkarni (1963) measured a mean of 0.25 ug/l in expired breath of nine "normal" people (range: 0.06 to 0.49 ug/l). Methanol is available in the diet from eating fresh fruits and vegetables or from drinking fruit juices (average of 140 mg/l; range: 12 to 640 mg/l) and fermented beverages (up to 1.5 g/l) (Francot and Geoffroy, 1956). More recently, aspartame, an artificial sweetener, has become a part of most diets. In the gut, aspartame hydrolyzes, and 10% of its molecule, by weight, becomes free methanol that is available for absorption (Stegink, 1984). According to recent estimates, excluding carbonated beverages, daily aspartame intake will average from 3 to 11 mg/kg, with the 99th percentile ingesting up to 34 mg/kg (i.e., 3.4 mg/kg methanol) (Stegink, 1984). Carbonated beverages contain about 555 mg aspartame per liter, which means that drinking a 12-ounce (354 cc) beverage is roughly equivalent to a methanol intake of 20 mg. In addition, methanol is generated metabolically by the action of a methyltransferase enzyme system. This system methylates acceptor proteins by the action of protein carboxyl methylase, and hydrolyzes protein-methyl esters (by the action of protein methyl esterase), which releases free methanol. (Gagnon and Heisler, 1979; Stegink et al, 1983). The relative contributions of diet and metabolism to the methanol body burden has not been established.

Formate is present in the blood at background levels that range from 3 to 19 mg/l (Baumann and Angerer, 1979; Stegink et al, 1981). Formic acid is a natural ingredient of various foods such as honey (2 to 200 mg/100 g), fruit syrups (65 to 163 mg/100 g), and roasted coffee (200 to 770 mg/100 g), and also is used as a preservative (FASEB, 1976). Formic acid also participates in several metabolic processes; for example, it is a product of the metabolic degradation of several amino acids, including histidine and tryptophan, and also serves as a precursor for a variety of macromolecules (FASEB, 1976; FPF, 1982). Perhaps most germane to the present discussion, formate is a metabolite of methanol and is metabolized in the folate pathway, as discussed in detail in Section IV. The relative contributions of diet, metabolism, and methanol breakdown to the formate body burden are unknown.

The reader should bear in mind that all projections of body burdens of methanol calculated in the following section of this report reflect only the environmental contribution. For purposes of evaluation and perspective, these contributions should be compared to background levels.

IV. METABOLISM OF METHANOL AND MECHANISMS OF TOXICITY

The nature of the dose-effect relationship for any substance is rooted in the mechanisms that govern that substance's uptake and processing in the body. The better the understanding of those mechanisms, the greater one's ability to project the potential consequences that may result from an environmental exposure of a specific magnitude and time course. Furthermore, elucidating the physiologic and biochemical pathways of action furthers the understanding of the determinants of inter-individual variability and, therefore, of individual sensitivity.

This section describes the fate of inhaled methanol and its metabolites. Its objective is to provide a mechanistic and quantitative basis for methanol's acute toxic syndrome, such that one may assess the potential of anticipated ambient exposures (Section II) to initiate known toxic processes. The exposures of concern in this report, in all likelihood, result in methanol doses well below those that produce the acute effects discussed in the previous section. The literature on metabolism and toxic mechanisms, although concerned primarily with the clinical (i.e., mostly acute) aspects of poisoning, is, nevertheless, highly relevant to the objectives of this report.

Unfortunately, there are no data with which to address directly the mechanisms that underlie methanol's chronic effects. However, because the chronic effects on record (Section III and Appendix II) bear a qualitative similarity to the well-studied acute effects, one may adopt a "working" assumption that acute and chronic effects share, to some extent, common pathways of action. Of course, the possibility that chronic exposure induces effects by means presently unknown is one that must, by definition, remain open.

Section III described the early developments that led to the recognition of the non-human primate as an appropriate model for acute human methanol toxicity. Despite the unique qualities of non-human primates, there are many important characteristics of methanol's uptake and metabolism that are common to all mammalian species, and these are covered first.

UPTAKE AND DISTRIBUTION

Regardless of its exposure route, methanol distributes readily and uniformly to all organs and tissues in direct relation to their water content (Yant and Schrenk, 1937). Methanol's distribution throughout the body is, therefore, ubiquitous. Blood methanol concentration, a parameter used frequently in the literature to characterize body burden of methanol, is, on the average equal to 83% of its aqueous concen-

tration. Urine, the other body fluid most commonly sampled, contains methanol concentrations 20 to 30% higher than blood (Yant and Schrenk, 1937; Leaf and Zatman, 1952).

The means of methanol's distribution explains that all exposure routes — oral, percutaneous, intra-peritoneal, and inhalation — are equivalent toxicologically. For ingestion and intra-peritoneal administration, the initial body burden is simply the amount of methanol given divided by body weight. For short-term inhalation, an upper-bound estimate of initial body burden assumes a total absorption of the inhaled vapor. This value is calculated as $(M_v \times \dot{V}_E \times t) / BW$, where M_v = vapor concentration, \dot{V}_E = ventilation rate, t = duration of exposure, BW = body weight. In reality, the absorption of inhaled methanol is less than 100%, with one reference reporting about 60% (Sedivec et al, 1981). An immediate application of this formula is to calculate the maximum initial body burden of methanol under an anticipated, worst-case, condition. Such a case might involve a 70 kg (BW) person, breathing at a rate of 20 m³/day (\dot{V}_E) (i.e., roughly twice resting ventilation), exposed to 200 mg/m³ (M_v) methanol vapor for 15 min (t) in a personal garage hot-soak situation (no ventilation, disabled canister). For 100% absorption, the resulting body burden is 0.6 mg/kg, which is at least 500 times lower than doses of acute clinical significance (i.e., greater than 0.3 g/kg). Table 6 lists the methanol body burdens that follow inhalation in several

Table 6. Methanol Body Burden for Selected Situations

Exposure Scenario	Condition	Added Body Burden of Methanol
Personal Garage Hot-Soak	200 mg/m ³ , 15 min, twice resting vent	0.6 mg/kg ^a
	100 mg/m ³ , 5 min, twice resting vent	0.1 mg/kg
	100 mg/m ³ , 5 min, resting ventilation	0.03 mg/kg
Self-Service Refueling	50 mg/m ³ , 4 min, twice resting vent	0.04 mg/kg
12 Ounce Diet Beverage	555 mg aspartame/l	0.3 mg/kg ^b
Dietary Intake of Aspartame (w/o diet beverages)	Normal Diet	0.3 - 1.1 mg/kg/day
"Background" Body Burden		0.5 mg/kg ^c

^a - assumes all inhaled methanol absorbed across respiratory epithelium; in all probability, less (appx 60% of inhaled) is absorbed (Sedivec et al, 1981).

^b - assumes all aspartame-derived methanol crosses gut mucosa instantaneously; because of the time required for hydrolysis and transport, peak measured levels reach 70-75% of the value in the table (Stegink et al, 1981).

^c - based on value of 0.73 mg/l of blood (Sedivec et al, 1981).

scenarios of interest and those that follow dietary intake of aspartame; the table also indicates the average background body burden of methanol.

CLEARANCE AND METABOLIC KINETICS

Following uptake and distribution, methanol clears from the body. Clearance proceeds with half-times of a day or more for high doses (greater than 1 g/kg) and about three hours for low doses (less than 0.1 g/kg) (Leaf and Zatman, 1952; Kane et al, 1968; Tephly and McMartin, 1984). Methanol is either excreted unchanged (direct excretion) in urine and exhaled breath, or it enters a metabolic pathway (in the liver), whose ultimate product is carbon dioxide. The metabolic process is of importance, because methanol's acute toxic properties (Section III) are linked to intermediate metabolites, not to the alcohol itself.

The time course of methanol's disappearance from the circulation reflects the combined action of both direct excretion and metabolism. A number of studies have been conducted in humans to examine for clearance of methanol from the circulation following low-level exposures.

Leaf and Zatman (1952) monitored methanol disappearance from the circulation of three human volunteers to whom 3, 5, and 7 milliliters (or 2.4, 4.0, and 5.6 grams) of methanol were administered orally (highest dose, 0.08 g/kg). Blood levels were reflected in urine samples collected every two hours for at least 12 hours. The results were consistent for all subjects at all doses: methanol disappearance obeyed first-order kinetics with a half-time of about 3 hours (i.e., every 3 hours, the concentration of methanol was halved). See the sidebar for a description of first-order kinetics.

Sedivec et al (1981) exposed four volunteers for eight hours to methanol vapors at concentrations of 102, 205, and 300 mg/m³. They analyzed urine samples through the exposure period and for the 18 hours that followed exposure. Urine levels of methanol were proportional to the vapor concentration throughout the observation period and, when exposure terminated, urinary methanol concentrations decreased exponentially with a half-life of roughly 2.5 to 3 hours.

Similar kinetics of methanol disappearance from blood were observed in human adults and infants to whom aspartame (10% methanol) was administered (Stegink et al, 1981; Stegink et al, 1983). The adult subjects received the equivalent of up to 20 mg/kg methanol and the infants, 10 mg/kg. Therefore, the clearance of methanol from the human circulation after body burdens as high as 80 mg/kg are achieved follows first-order kinetics with a half-time ($T_{1/2}$) of about 2.5 to 3 hours; the rate constant for total clearance, k_T , is therefore $0.693/T_{1/2}$, or between 0.23 and 0.28 hr⁻¹.

Several studies have examined the percent of methanol that is excreted as CO₂. This value reflects the fraction of the initial dose of methanol that clears by the metabolic route. The available data suggest that, following a low dose of methanol (2 mg/kg) to non-human primates (rhesus monkeys) and rats (CD-1), as much as 90% is metabolized (Opperman, 1984). Because methanol metabolism is a saturable process (see below), one expects its efficiency to decrease with larger doses. However, Eells et al (1983) showed that even after a very high

dose (1 g/kg) of radiolabeled methanol to cynomolgus monkeys, 78% of the activity recovered within 24 hours was as exhaled CO₂.

SIDEBAR

FIRST-ORDER KINETICS

A first-order clearance or metabolic process is one that proceeds at a rate proportional to the concentration of the substance undergoing that process. Expressed mathematically, $v_1 = k_1 x(t)$

where $x(t)$ = concentration of substance x at time t

v_1 = rate at which x is processed through pathway #1

k_1 = clearance constant for pathway #1

For any pathway that obeys first-order kinetics,

$$x(t) = x_0 e^{-k_1 t},$$

where x_0 = concentration of x at $t = 0$

and, in addition, $T_{1/2} = 0.693/k_1$, the interval required to halve the concentration of x through pathway 1 alone.

If a second first-order pathway with clearance constant k_2 is operative simultaneously, then total clearance is described by $x(t) = x_0 e^{-(k_1 + k_2)t}$. Further, pathway #1 is responsible for $k_1/(k_1 + k_2)$ of the total cleared and #2 for $k_2/(k_1 + k_2)$ of the total. For three pathways, the rate constant is $k_1 + k_2 + k_3$, pathway #1 metabolizes $k_1/(k_1 + k_2 + k_3)$ of the total, and so on.

MICHAELIS-MENTEN KINETICS

In a reaction that obeys Michaelis-Menten kinetics, v , the rate of that reaction (e.g., metabolic conversion), is related to the concentration of the reactant (in this case, methanol) as follows:

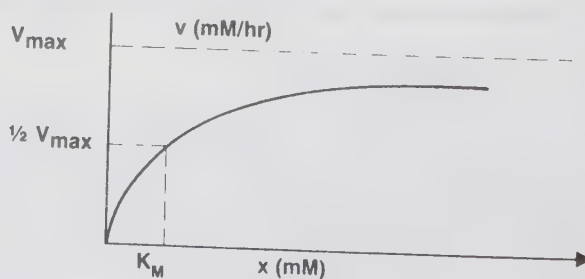
$$v = V_{\max} \frac{x}{K_M + x}$$

where, V_{\max} = maximum velocity of reaction as x approaches infinity and,

K_M = the Michaelis constant, which defines the concentration at which

$$v = \frac{1}{2} V_{\max}$$

The relationship, drawn in the figure, reflects that at concentrations large compared to K_M , the reaction approaches saturation. In other words, it operates with nearly zero-order kinetics, which means that v is nearly independent of x . At low concentrations that are small compared to K_M , $v = (V_{\max}/K_M)x$. Such reactions are first-order because the reaction velocity is related almost linearly to concentration or $v = kx$ and $k = V_{\max}/K_M$.



MECHANISMS OF CLEARANCE

The discussion that follows develops in greater detail the mechanisms that account for metabolism's dominant role in methanol clearance. This analysis will enable a sharper assessment of the potential for health effects under expected exposure conditions.

The observations that total clearance of methanol from body water proceeds with first-order kinetics at low doses (less than 0.1 g/kg) justifies the supposition that each separate pathway also obeys first-order kinetics, and is, thus, characterized uniquely by its own rate constant (see sidebar). If such is the case, k_T , the rate constant for total clearance, equals the sum of rate constants for all pathways; further, the fractional contribution of each pathway to clearance is equal to its rate constant divided by the sum of constants.

Each of the three major clearance pathways that were identified earlier may be assigned a rate constant: k_r represents direct renal excretion of methanol; k_p represents direct pulmonary excretion of methanol; and k_m represents metabolic clearance. Expressed mathematically, the preceding states that, if all pathways obey first-order kinetics, then, $k_T = k_r + k_p + k_m$, and that the fractional contribution of each pathway, for example, metabolism, is k_m/k_T . These pathways are displayed schematically in Figure 2. The figure indicates with a dotted line that, in addition to CO_2 , the metabolic pathway generates formate, which is detectable in urine. Formate is the toxic metabolite apparently responsible for methanol's acute toxic syndrome in humans and primates. However, at the low doses of concern in this report, formate, as a metabolic end product, is excreted in quantities that are negligible compared to CO_2 . Thus, for the analysis presented in the discussion that follows, CO_2 excretion represents the quantitative metabolism of methanol. The principles governing formate generation are explored, in depth, later in this section.

In the following analysis, rate constants for each pathway are derived on the basis of physiologic or kinetic principles, and, to test their validity, they are compared to empirical data:

(1) Direct renal excretion of methanol: The kidney apparently exerts no active control over the urinary concentration of methanol. Consequently, the methanol content of urine that enters the bladder maintains the aqueous concentration of methanol in the blood that entered the kidney (Yant and Schrenk, 1937; Leaf and Zatman, 1952; Sedivec et al., 1981). Thus, the rate at which methanol clears into the urine is directly proportional to its blood level, which satisfies the condition for first-order kinetics.

Under these conditions, k_r , the rate constant for direct renal excretion of methanol, equals the rate of urine formation divided by the total volume of body water. A "typical" 70 kg person contains roughly 42 liters of water (60%) and produces on the average 60 milliliters (ml) of urine per hour (0.86 ml/hr/kg). Therefore, one may project a k_r of 0.0014 hr^{-1} ; half-time for this pathway is simply $0.693/k_r$ (20 days). This simple formulation allows one to predict that renal processes account for k_r/k_T of all the methanol cleared. Since k_T was estimated earlier as 0.23, the renal contribution to the total is about 0.6%.

Leaf and Zatman (1952) measured urinary excretion of methanol from three human subjects after they ingested a dose of 3.2 g of methanol (avg 0.05 g/kg). These investigators determined that at 12 hours post-ingestion (when more than 90% of a methanol dose has cleared the circulation), an average of 0.76% of the initial dose was excreted in the urine. Jacobsen et al (1983) studied renal clearance of methanol in a 65 kg victim of methanol poisoning who was undergoing treatment, which included diuresis of 7.0 liters over a 24 hour period, i.e., a urine production of 4.2 ml/hr/kg; as mentioned above, urine formation in a "normal" 70 kg person proceeds at around 60 ml/hr, or 0.86 ml/hr/kg. Renal clearance of methanol in the patient was measured as 5.7 ml/min (0.34 l/hr). (Clearance expressed in this manner has only a hypothetical meaning — it is as if each minute, 5.7 ml of blood were totally cleared of methanol.) Assuming the patient had a body water content of 60%, and a blood water content of 83%, the calculated

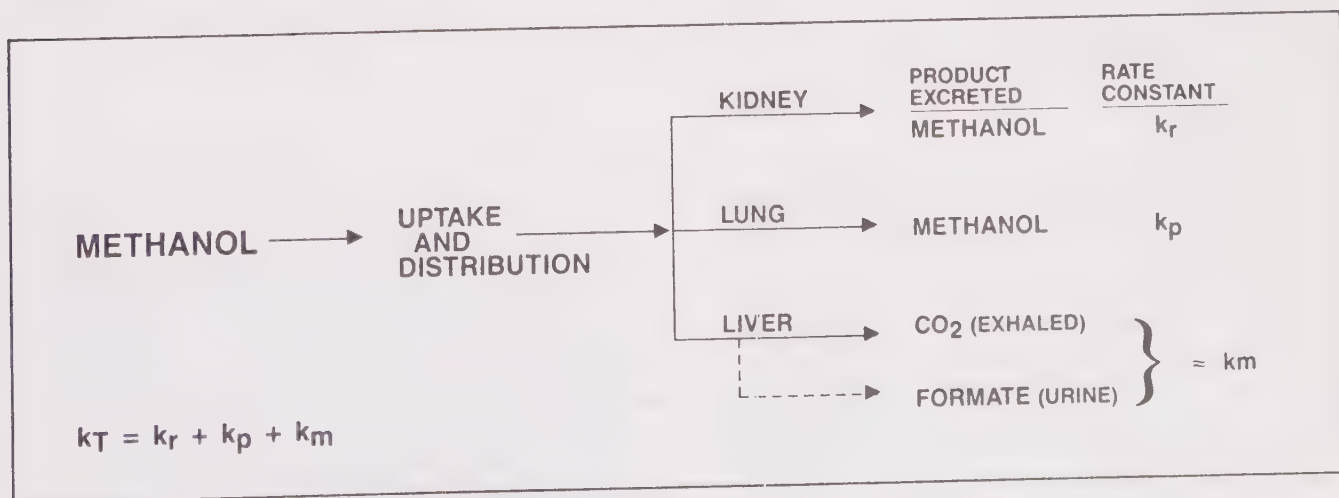


Figure 2. Schematic Diagram of Clearance Pathways for Methanol Following Uptake and Distribution Throughout the Body.

rate constant for renal clearance of methanol during diuresis is, $(0.34 \text{ l/hr} \times 0.83)/(65 \text{ kg} \times 0.6 \text{ l/kg}) = 0.007 \text{ hr}^{-1}$.

The value just calculated was obtained from the study of a patient treated with diuresis. To obtain a renal clearance constant (k_r) for a person who is producing "normal" quantities of urine requires multiplying the clearance value just calculated by the ratio of "normal" urine production (0.86 ml/hr/kg) to urine production in the patient (4.2 ml/hr/kg), which yields a k_r of 0.0014 hr^{-1} . On the basis of these data and calculations, the above estimate of k_r seems reasonable.

(2) Direct pulmonary excretion of methanol: In the lung, a small fraction of blood-borne methanol diffuses passively from the pulmonary capillaries to the alveoli and is exhaled. The amount of methanol that crosses the blood-air barrier is directly proportional to its blood concentration — i.e., first-order kinetics — and is governed by its blood-air partition ratio; this parameter describes the relative content, at equilibrium, of a substance in each of two phases in contact.

For a first-order process, the rate of pulmonary excretion of methanol = $k_p \times$ blood methanol concentration. The rate constant, k_p , may be approximated by $V_A/(\text{PR} \times \text{bw})$ where,

V_A = alveolar ventilation, approx 500 liters/hour during moderate activity for a typical 70 kg person.

bw = body water, 42 liters for same person.

PR = methanol's blood-air partition ratio, approximately 2,000 (Harger et al, 1950).

Plugging in the above values yields a value for k_p of 0.006 hr^{-1} (and therefore, a 5 day half-time). This derivation of k_p 's hypothetical value is based, for purposes of simplification, on a constant alveolar ventilation. However, altering ventilation will affect the rate at which methanol is exhaled.

Jacobsen et al (1983) also studied pulmonary excretion of methanol in the 65 kg patient described above. The patient, though admitted with methanol poisoning, was not hyper-ventilating. These investigators calculated a pulmonary clearance rate of 5.6 ml of blood per minute (0.34 l/hr) (i.e., each minute, 5.6 ml of blood is hypothetically cleared totally of methanol). Assuming the patient had a body water content of 60% and a blood water content of 83%, the calculated rate constant k_p equals $(0.34 \text{ l/hr} \times 0.83)/(65 \text{ kg} \times 0.6 \text{ l/kg}) = 0.007 \text{ hr}^{-1}$. This value and the theoretical estimate for k_p (0.006 hr^{-1}) are quite consistent.

(3) Metabolic clearance: In contrast to direct renal and pulmonary excretion, the metabolic conversion of methanol to carbon dioxide is not linear with concentration. In the 1960s, Tephly and colleagues demonstrated, in both rats (Holtzman) and rhesus monkeys, that in vivo metabolism of methanol to CO_2 obeys Michaelis-Menten kinetics (see sidebar) and, further, that the metabolic kinetics for both species are very similar (Table 7) (Tephly et al, 1964; Makar et al, 1968). Briefly, this means that with rising blood concentrations of methanol, the metabolic pathway demonstrates saturability, and approaches a maximal conversion rate that it is unable to exceed. At saturation, the pathway demonstrates zero-order

kinetics, which means the metabolic conversion rate is independent of concentration. However, at low concentrations — much lower than the Michaelis constant, K_M — processes that obey Michaelis-Menten kinetics behave as if they are first-order processes, with a rate constant, k_m of V_{\max}/K_M (explained in sidebar). Subsequent time-course observations (Noker et al, 1980) of methanol clearance from the blood of cynomolgus monkeys given 2 g/kg of methanol provide data that are consistent with those on rhesus monkeys shown in Table 7; specifically, Noker et al (1980) recorded a zero-order clearance of about 50 mg/kg/hr, and their graphic data display a transition from zero to first-order kinetics at around 10 millimoles of methanol per liter of blood or 12 mM aqueous concentration.

Table 7. "In Vivo" Michaelis-Menten Constants for the Metabolic Conversion of C^{14} -Methanol to C^{14}O_2

Species	V_{\max} (mg/kg/hr)	K_m (mM)
Non-human Primates ¹ (rhesus monkeys)	48	8.7
Rats ²	30	7.0

¹ - Makar et al, 1968

² - Tephly et al, 1964

As estimated earlier in this section, a worst-case single exposure (200 mg/ m_3 methanol for 15 minutes) produces a methanol body burden of less than 1 mg/kg. For an individual with 60% body water, this upper level burden corresponds to 0.05 millimoles of methanol per liter (mM). This concentration is less than 1% of the K_M for monkeys (roughly 10 mM), which reinforces the notion that, for environmental exposures of interest in this report, metabolism is operating in a first-order domain. To calculate k_m requires transforming V_{\max} into units of mM/hr. Makar et al (1968) used carbon-14 labeled methanol (MW 34), and estimated body water of rhesus monkeys at 70% of their weight. Thus, based on the values for rhesus monkeys in Table 7,

$$k_m = \frac{V_{\max}}{K_M} = \frac{1}{8.7} \times \frac{48}{(34)(0.7)} = 0.23 \text{ hr}^{-1}$$

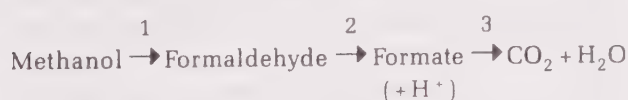
The metabolic rate constant derived here is clearly much greater than the sum of constants for both direct renal (0.0014 hr^{-1}) and direct pulmonary (0.0060 hr^{-1}) excretion, which explains, from a kinetic viewpoint, why metabolism is expected to dominate the clearance of methanol from body water. As indicated earlier, this dominance has been confirmed in animal experiments. Table 8 summarizes the preceding analyses, and demonstrates that the clearance patterns of methanol observed empirically are consistent with those one would project using basic physiologic principles and Michaelis-Menten constants derived from studies of non-human primates.

Table 8. Contribution of Renal, Pulmonary, and Metabolic Pathways to the Overall Clearance of Low Doses of Methanol (< 0.1 g/kg) : Comparison of Observed and Derived Rate Constants

Clearance Pathway	Rate Constant k (hr ⁻¹)	Derived Values		Exptl/Clin Observations	Source
		Percent Clearance	Source		
Direct Renal	0.0014	0.6	Basic Considerations	0.76% of initial dose excreted in urine by 12 hours post-ingestion k _r of 0.0014 hr ⁻¹ for "normal" derived from patient on diuresis	Leaf and Zatman, 1952 Jacobsen et al, 1983
Direct Pulmonary	0.0060	2.5	Basic Considerations (Partition ratio from Harger et al, 1950)	k _p of 0.007 hr ⁻¹ derived from patient data	Jacobsen et al, 1983
Metabolic	0.23	96.9	Makar et al, 1968	90% or more of dose excreted as CO ₂	Opperman, 1984
Total	0.24	100.0		half-time of approximately 2.5 to 3 hours observed in humans, i.e., k _T = 0.23-0.28	Leaf and Zatman, 1952 Sedivec et al, 1981 Stegink et al, 1981 Stegink et al, 1983

TOXIC MECHANISMS AND SPECIES SENSITIVITY

The above discussion dealt with metabolism as if it proceeded as a "black-box" process with input (methanol) and output (CO₂). This subsection probes the workings of that "black box," and describes the biochemical events within the metabolic pathway that trigger methanol's toxic response, and discusses the major factors that influence species and individual susceptibility. The mechanisms discussed are associated directly with acute effects, as laboratory experiments and clinical analyses have amply demonstrated, but they probably contribute to chronic effects as well. In the liver, the sequence of intermediate metabolites that lead from methanol to carbon dioxide is the same in all mammalian species studied (Tephly and McMartin, 1984):



Although the sequence of metabolites is identical for all species, there are interspecies differences with respect to the details of the reactions. Several of these differences bear directly on sensitivity to methanol and several appear not to. Appendix VII, excerpted from Tephly and McMartin (1984), describes in depth the details of methanol metabolism in both non-human primates and non-primates. The discussion that follows briefly reviews the entire metabolic process, but deals mainly with the aspects of metabolism related to toxicity.

The toxic properties of methanol, and the basis of species susceptibility, are rooted in the factors that govern the relative rates of formic acid (formate + H⁺) generation (steps 1 and 2) and formate oxidation to CO₂ (step 3). In short, the toxic

syndrome sets in if formate generation continues at a rate that exceeds its rate of removal; the latter is a mainly a function of metabolism to CO₂, as renal excretion of formate plays a minor role. This imbalance, if protracted, leads to an accumulation of formate coupled, eventually, to an uncompensated metabolic acidosis. The acidosis, if untreated, can prove lethal; however, even at physiologic pH, formate is associated with ocular toxicity. Rats dispose of formate efficiently at any dose and thus escape toxicity, whereas, at sufficiently high doses, humans and non-human primates accumulate toxic metabolites and do not fare as well. The key to species differences lies in step 3 and, as explained below, it is directly related to the maximum rate at which the liver oxidizes formate.

Step 1: The first step in the metabolic sequence, in which methanol is oxidized to formaldehyde, reflects the major route of methanol clearance from the circulation (see previous discussion). In non-human primates, alcohol dehydrogenase mediates this initial step (rhesus monkeys, McMartin et al, 1975); in rats, a catalase-peroxidative system is primarily responsible (Tephly et al, 1964 - Holtzman strain; Makar and Mannering, 1968 - Sprague-Dawley). Despite this difference, the initial metabolic step proceeds at similar rates in non-human primates and rats (Tephly et al, 1964; Makar et al, 1968; Tephly and McMartin, 1984). The values for V_{max} in Table 7 represent, fairly well, the maximal clearance rates observed in both species, although several studies in non-human primates have displayed even higher rates (up to 62 mg/kg/hr) (Bartlett, 1950; McMartin et al, 1975; Noker et al, 1980; Eells et al, 1983). The rate at which methanol disappears from the circulation and enters its metabolic process is independent

of "downstream" manipulations that either stimulate or slow the oxidation of formate (step 3) (Eells et al, 1983).

Step 2: The second metabolic step converts formaldehyde to formic acid, which, in turn, dissociates to formate and a hydrogen ion. The step occurs as a two-reaction process, the second of which is irreversible. The first reaction, in which formaldehyde is oxidized to S-formylglutathione, requires reduced glutathione (GSH), and is mediated by an NAD-dependent formaldehyde dehydrogenase. In the second reaction, thiolase catalyzes the conversion of S-formylglutathione to formic acid (Tephly and McMartin, 1984).

The nature of formaldehyde oxidation is important because formaldehyde itself is potentially toxic, and several "modern" references ascribe methanol's ocular toxicity to it (Loomis, 1968; Casarett and Doull, 1975). Formaldehyde has not, however, been detected in body fluids or tissues following toxic methanol exposures (Makar and Tephly, 1977; McMartin et al, 1977b; McMartin et al, 1979). Furthermore, its clearance from the bloodstream following intravenous infusion occurs with a half-life of between one and one-and-a-half minutes, and is followed by an immediate and corresponding rise in blood formate (Malorny et al, 1965; Rietbrock, 1965; McMartin et al, 1979). The possibility that formaldehyde generated in the liver is active in the optic tract seems unlikely. Martin-Amat et al (1977) have demonstrated, moreover, that formate alone given intravenously to rhesus monkeys induces the same ocular toxicity that is characteristic of methanol poisoning, with no sign of formaldehyde present.

Thus, evidence supporting a role for formaldehyde in methanol toxicity is still lacking. The possibility that formaldehyde is generated locally in the retina or proximal optic nerve has not yet been ruled out, and may merit some further attention. However, the present focus on formate as the principal toxic agent appears justified.

Step 3: In both non-human primates and rats, a folate-dependent pathway in the liver is responsible for the reactions that metabolize formate (step 3). Figure 3 shows the major aspects of this pathway. Formate first forms a complex with tetrahydrofolate (THF) that is sequentially converted to 10-formyl-THF (by formyl-THF synthetase) and then to carbon dioxide (by formyl-THF dehydrogenase). THF is derived from folic acid in the diet and also is regenerated in the folate pathway.

Although the folate pathway metabolizes formate in both species, rats use the pathway more efficiently, a fact that lies at the heart of species sensitivity to methanol. Both formate clearance from the blood and its metabolism to CO₂ proceed about 2 to 2½ times faster in rats than in non-human primates. For example, Clay et al (1975) showed that, following its intravenous infusion at doses lower than 100 mg/kg (2.2 mmole/kg), formate clears from rats with a half-time of 12 minutes, and from non-human primates (pigtail macaque) with a half-time of 31 minutes, and that the respective half-times decrease with increasing dose in both species, which indicates that formate metabolism is a saturable process; these

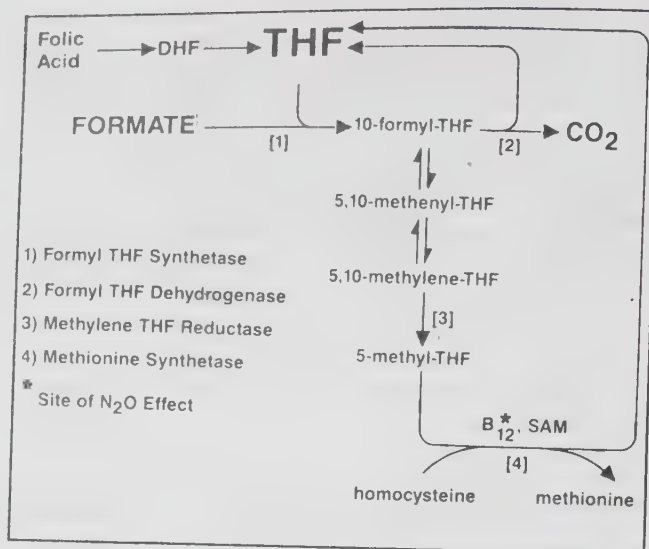


Figure 3: Metabolism of formate by folate-dependent reactions. DHF, dihydrofolic acid; THF, tetrahydrofolic acid; SAM, S-adenosylmethionine. Reaction 1 is catalyzed by formyl-THF synthetase and requires prior activation of formate with ATP. Reaction 2 involves [¹⁰N] formyl-THF dehydrogenase and utilizes NADP as a hydrogen acceptor. Reaction 3 is catalyzed by 5,10-methylene-THF reductase and is essentially irreversible. Reaction 4 is catalyzed by 5-methyl-THF homocysteine methyltransferase (methionine synthetase) and requires catalytic amounts of vitamin B₁₂ and SAM.

Source: Eells et al., 1981

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findings are consistent with previous research (Malorny, 1969a; Rietbrock, 1969). Specifically, in the study by Clay et al (1975), half-times in pigtail monkeys were 51 minutes at 470 mg/kg, 49 minutes at 285 mg/kg, 46 minutes at 200 mg/kg and 31 minutes at both 72 and 50 mg/kg; at 670 mg/kg, formate half-time in rats was 23 minutes. However, following high doses of methanol (4 g/kg in pigtail monkeys, 6 g/kg in Sprague-Dawley rats) formate accumulates appreciably only in monkeys, with only a subtle increase seen in rats (Figure 4) (Clay et al, 1975). On the basis of relative metabolic efficiencies, one may have expected rats to accumulate 40 to 50% of the formate measured in non-human primates.

The apparent reason why rats do not accumulate formate noticeably at any dose of methanol is that the maximal rate of formate oxidation in rats (1.6 mmole/kg/hr) exceeds the maximal rate that steps 1 and 2 supply substrate (from Table 7, 30 mg/kg/hr, equivalent to 0.9 mmole/kg/hr). On the other hand, in non-human primates, the "upstream" processes are able, with high enough methanol concentrations, to feed formate to the folate pathway at a rate (1.4 to 1.8 mmole/kg/hr) that exceeds the pathway's metabolic capacity (0.75 mmole/kg/hr). The "spillover" accumulates in the circulation which, if protracted, leads to the toxic consequences of methanol poisoning. These relationships are shown in Figure 5. At low doses of methanol, the process that clears methanol from the bloodstream and generates formate (3 hour half-time) is considerably less efficient than the process that clears formate (half-hour half-time in primates). Therefore, at low methanol expo-

tures, formate is not expected to accumulate to any major degree. Baumann and Angerer (1979) report that 20 workers exposed to an average of 140 mg/m³ methanol for an 8-hour work day registered an increased blood formate of 0.1 mM at the end of their work-shift (background for humans ranges from 0.07 to 0.4 mM — see Section III). Urinary excretion of formate, though detectable following both human and animal methanol exposures, plays a minor role relative to metabolism in the overall clearance of formate.

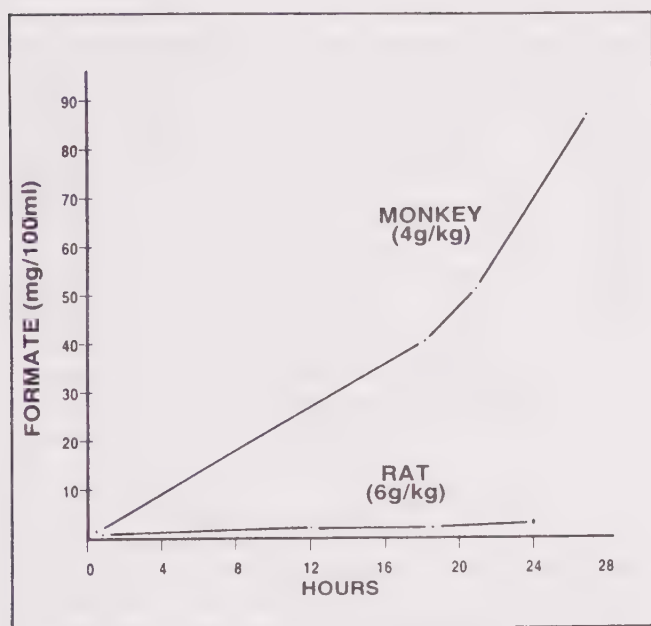


Figure 4: Blood formate concentrations in the monkey and rat after administration of methanol. Methanol was administered as a 25% solution in saline to the monkey (4 g/kg, ip) and the rat (6 g/kg, ip). Blood specimens were drawn at the indicated times after methanol administration.

Source: Clay et al, 1975

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The efficiency of formate metabolism is linked strongly to the hepatic concentration of tetrahydrofolate (THF), the molecule that initially complexes with formate. The factors, in turn, that control THF levels involve both the dietary level of folate and the dynamic equilibrium within the biochemical loop that regenerates THF (Figure 3).

In the 1970s, Tephly and colleagues studied the effects of dietary folate on species susceptibility to methanol toxicity. Makar and Tephly (1976) fed rats (Sprague-Dawley) a folate deficient diet, and observed that these subjects suffer the effects of a methanol dose (4 g/kg) that such animals ordinarily tolerate well when maintained on a control diet. (The folate-deficient period, though not specified in Makar and Tephly (1976), was probably similar to the 6 to 8 week period used by Palese and Tephly (1975).) Specifically, folate-deficient rats became acidotic and accumulated formate (but not formaldehyde) in the same manner observed previously in non-human primates (Makar and Tephly, 1976, 1977). The key to methanol toxicity in folate-deficient rats is a diminished

capacity to oxidize formate (Palese and Tephly, 1975; Makar and Tephly, 1977). On the other hand, cynomolgus monkeys treated with folate or a folate derivative (5-formyl-THF), and given 2 g/kg of methanol, maintained normal blood pH associated with only a modest rise in blood formate (Noker et al, 1980); control monkeys experienced a larger rise in formate and became temporarily acidotic. As one would expect, folate-deficient monkeys displayed even greater susceptibility to methanol than their normally fed counterparts (McMartin et al, 1977a). In all of these cases, the effect of dietary folate on formate metabolism was the direct link to altered sensitivity to methanol; slowing formate metabolism induced a methanol-sensitive state.

The dependence of formate oxidation on endogenous folate regeneration has been demonstrated in both rats and non-human primates. Using both species, Eells and colleagues performed a series of experiments in which the folate feedback loop was opened pharmacologically and the animals then were challenged with either formate or methanol (Eells et al, 1981; Eells et al, 1982; Eells et al, 1983). The agent used to poison the loop was nitrous oxide (N₂O), an anesthetic gas, which, among its actions, blocks methionine synthetase (Figure 3, Step 4) by inactivating vitamin B₁₂, the enzyme's cofactor. Nitrous oxide treatment slowed formate metabolism and heightened methanol sensitivity. With N₂O, rats (Sprague-Dawley) given 4g/kg methanol and monkeys (cynomolgus), given 1 g/kg became acidotic, but animals given methanol without N₂O maintained normal blood pH.

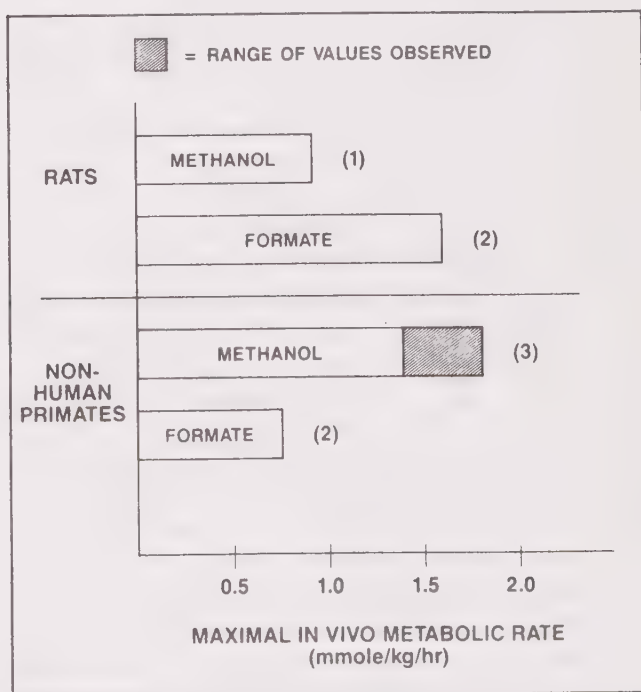


Figure 5: Maximal In Vivo Metabolic Rates for Methanol and Formate in the Rat and Non-human Primate

Source: (1) Tephly et al, 1964

(2) McMartin et al, 1977a

(3) Makar et al, 1968, McMartin et al, 1975, Noker et al, 1980, Eells et al, 1983

Eells et al (1982) further showed, using Sprague-Dawley rats, that the critical variable in these experiments was the concentration of hepatic THF. The concentration of total hepatic folate remained unaffected by N_2O treatment. The extent to which N_2O slowed formate oxidation was linked directly to the extent that N_2O decreased hepatic levels of THF. When plotted against each other, formate oxidation and hepatic THF correlated very closely ($r = 0.89$). Nitrous oxide thus acts to upset the dynamic equilibrium of the folate regenerative pathway so as to increase methylated and formylated folates at the expense of THF.

Subsequent research has focused on the comparative aspects of folate biochemistry among rats, monkeys, and humans. Black et al (1985) studied rats (Sprague-Dawley) and non-human primates (cynomolgus monkeys) and compared the levels of folate intermediates and the activities of folate-dependent enzymes in the livers of both species. Though total folate content of the two was practically identical, the THF concentration in monkey livers was 59% of the value in rats. This lower level of THF in non-human primates is consistent with their heightened sensitivity to methanol, and the ratio of THF levels in the two species is similar to the ratio of their maximal rates of formate oxidation (47%). The authors describe several species differences in folate-dependent enzyme activities that may contribute to different states of dynamic equilibrium in the folate regenerative pathway, whereby the species-specific balance of THF to total folate favors the rat's natural resistance to methanol. The authors note that the equivalence of total folate in both species "suggests that monkeys do not have a deficit in the dietary or hepatic uptake of folates, as compared to rats."

More recently, Johlin et al (1986) reported that human levels of total hepatic folate and THF were 60% and 50% of the respective levels in rat liver. Research is continuing to unravel the intricacies of the folate pathway and its relationship with methanol toxicity.

SEQUENCE OF ACUTE TOXICITY: ACIDOSIS AND OCULAR TOXICITY

The establishment and refinement of the non-human primate model has enabled researchers to both characterize carefully the sequence of events that follow large single doses of methanol, and to propose mechanisms of action. Figure 6 from Tephly (1977) illustrates the time course for several key blood parameters that follows a lethal (3 g/kg) dose of methanol. As shown, in the symptomless latent period, formate accumulates and homeostatic mechanisms compensate for the increasing acid load. Between 12 and 16 hours post-ingestion, homeostatic reserve is exhausted, and the animal enters an acidosis from which it does not recover. Through the latter phase, the blood level of formate (and perhaps of other organic ions such as lactate) continues to increase. With lower doses (0.5 g/kg and 2.0 g/kg) formate and acid start to accumulate, but, with a continuously diminishing supply of methanol in the bloodstream, the critical blood parameters

(formate, pH) revert to normal values (Figure 7). Presumably, at these lower doses, the initial steps that oxidize methanol generate formate in excess of the folate pathway's formate-metabolizing capacity. However, as methanol clearance continues, and its blood concentration drops, the rate of formate generation drops below the folate pathway's capacity, which allows both the throughput of formate and the excess blood formate to be metabolized, and results in a return to normal. For lethal doses, formate generation continues in excess of the formate-metabolizing capacity into the terminal state.

Non-human primates and humans that survive the lethal effects of methanol are still at considerable risk of developing damage to the visual system. In 1977, Martin-Amat et al introduced a non-human primate model (rhesus monkeys) for sustaining methanol toxicity at a sub-lethal level to allow full expression of the ocular toxicity to develop; acidosis and elevated formate in blood (and cerebrospinal fluid) characterized this sustained toxic period. Using this model, Hayreh et al (1977) noted ophthalmoscopic changes, consisting primarily of hyperemia and edema of the optic disc, between 43 and 171 hours from the start of ingestion. Except for some engorgement of the retinal veins in the later phase of edema, the retinal vasculature remained normal throughout the observation period. Optic disk edema was the only sign of angiographic abnormality in the entire retina. In addition, the monkeys' pupils were dilated and unresponsive to light, as with human intoxication.

Baumbach et al (1977) further explored the injury to the visual system using light and electron microscopic examinations of tissue from the same animals that Hayreh et al studied. According to their observations, the primary sites of injury are the optic nerve head and the intraorbital portion of the optic nerve (i.e., the site where the retinal nerve fibers converge and become a nerve bundle); the retina itself is undisturbed. They state, "The morphological alterations seen in the optic nerve and optic nerve head in methyl alcohol-poisoned rhesus monkeys can be separated into two categories — alterations within axons and alterations of glial cells. Alterations seen in axons include mitochondrial swelling and clustering, neurotubular disruption, the formation of vesicles, and increased density of amorphous proteins, and axonal enlargement. Glial cell alterations include astrocytic swelling and swelling of the oligodendroglial cytoplasm in contact with the optic nerve axon."

The precise mechanisms of this injury are not yet clear, but several investigators have suggested a sequence that involves the interaction of formate-induced metabolic inhibition with the observed compression and swelling of optic nerve fibers. The initiating event is believed to involve formate's action as a metabolic poison, specifically as an inhibitor of cytochrome oxidase. This protein is located in the mitochondria and is the last in a chain of cytochromes and other molecules that transfer electrons sequentially to molecular oxygen. In so doing, they generate adenosine triphosphate (ATP), the cell's basic molecular form of energy. Formate inhibits cytochrome

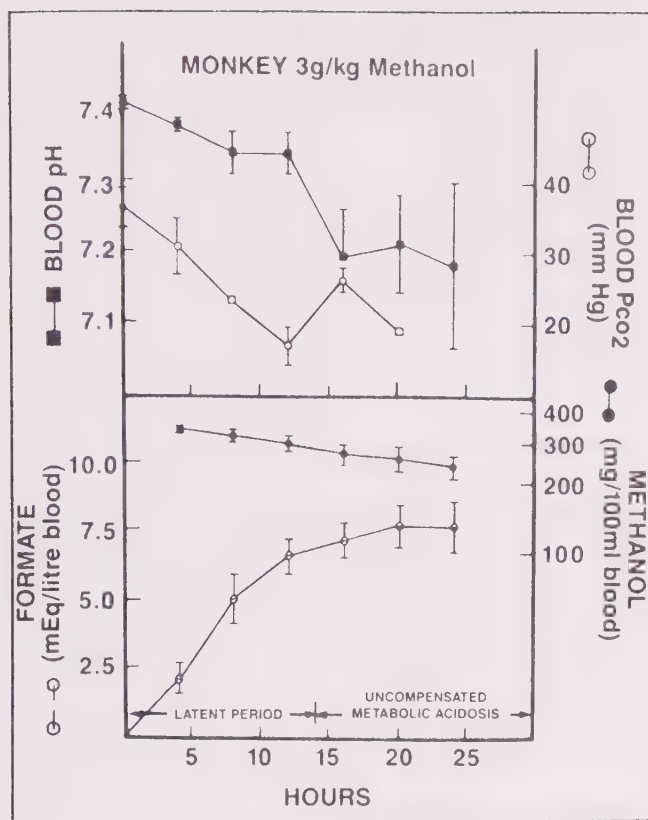


Figure 6: Metabolic acidosis, formate accumulation, and methanol metabolism in the monkey. Each point represents the mean \pm SEM of at least three animals.

Source: Tephly, 1977

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oxidase with a K_i (the concentration that achieves 50% cytochrome inhibition) of between 5 and 30 mM, which overlaps the range of formate concentrations observed in acute methanol intoxication both experimentally and clinically (Nicholls 1975,1976). Furthermore, as pH decreases, inhibition increases, which suggests that the active species is undissociated formic acid (described further below).

The myelin sheath (or white matter) of the optic nerve is comprised of cells that normally have low reserves of cytochrome oxidase, owing to their low metabolic requirement, and thus may be particularly sensitive to formate-induced metabolic inhibition. At the point where the optic nerve bundle forms, formate has access to the nerve from both the choroid circulation behind the retina and from the cerebrospinal fluid. [Martin-Amat et al (1977) observed formate accumulation in the cerebrospinal fluid of rhesus monkeys given large methanol doses; the CSF formate levels were similar to those in the blood.] If sufficiently damaged from metabolic inhibition, the cells of the white matter (oligodendroglial cells) may swell and, in essence, form small "cuffs" around individual nerve fibers, which themselves may then exhibit a compression-type injury in the form of swelling and edema proximal to the cuff in the optic nerve head. Such compression on the fiber will slow the transport of proteins, neurotubules, and mitochondria from the fiber's cell body (located

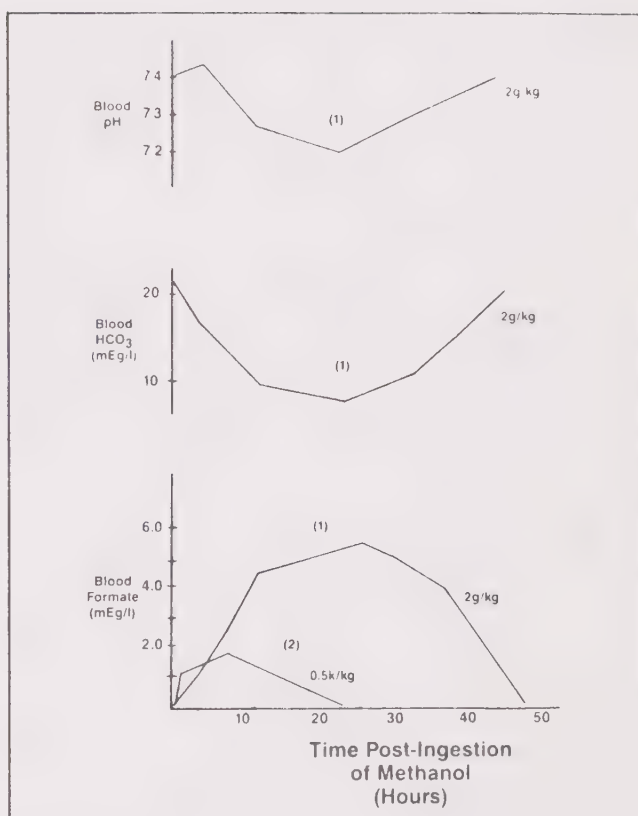


Figure 7: Blood levels of bicarbonate and formate and blood pH following oral administration of methanol to cynomolgus monkeys

Adapted from: (1) Noker et al, 1980

(2) McMartin et al, 1977a

in the retina — the retinal ganglion cell) to the fiber axoplasm which renders the fiber increasingly deficient in essentials and, thus, susceptible to formate-induced injury. Once the nerve fiber is no longer able to sustain the metabolic energy to conduct electrical impulses, visual decrements will occur. This schema for the progression of toxic events is consistent with the morphological evidence presented by Hayreh et al (1977) and Baumbach et al (1977), who used the non-human primate model for methanol-induced ocular toxicity.

The onset of acidosis may serve to accelerate the sequence just described. As mentioned, the inhibition of cytochrome oxidase increases with decreasing pH, which suggests that undissociated formic acid is the active inhibitor of cytochrome oxidase. The Henderson-Hasselbalch equation predicts that with a pH drop of 0.3, which is commonly observed in methanol poisoning, the concentration of undissociated formic acid (pK_a 3.8) doubles. Thus, acidosis may potentiate the biochemical inhibition of cellular respiration, and speed the onset of cellular injury. Also, the acidosis as it progresses will begin to induce circulatory failure, which leads to tissue hypoxia and lactate production, both of which further increase the acid load, increasing undissociated formic acid, etc. This cycle, termed "circulus hypoxicus" (Figure 8) by Jacobsen and McMartin (1986), may hasten the end stage consequences of methanol poisoning. While acidosis may accelerate formate

toxicity. Martin-Amat et al (1978) showed that, even at physiologic pH, formate is capable of inducing the same ocular toxicity in non-human primates (rhesus monkeys) as is observed in methanol poisoning. Formate maintained between 12 and 34 mEq/l (equivalent to mM) in the blood for 25 to 50 hours inhibited the pupillary response and caused optic disc edema.

ACCUMULATION OF FORMATE IN HUMANS: HIGH AND LOW DOSES

Large increases in circulating formate are linked firmly to the acute toxic manifestations that result from large doses of methanol (approximately 0.3 g/kg and above). As discussed earlier in this section, one would expect a sharp increase in formate accumulation to occur with saturation of the folate pathway, which metabolizes formate. Applying formulas and values used previously in this report, and assuming that non-human primates adequately model humans, one may calculate the approximate dose of methanol that achieves this saturation. Assume (1) the maximal rate of methanol metabolism (V_{max}) in humans is equivalent to the value given in Table 7 for non-human primates, 48 mg C^{14} -methanol/kg/hr (or 2.0 mM/hr for a non-human primate that consists of 70% water), (2) the maximal rate of formate oxidation is the same for both species, 35 mg C^{14} -formate/kg/hr or 1.1 mM/hr, (3) the Michaelis constant for methanol metabolism in humans is the same as in non-human primates, 8.7 mM, and (4) formate is relatively evenly distributed through body water, a reasonable assumption based on the determination by Jacobsen et al (1983) of formate's volume of distribution (0.5 liters/kg). Then using the Michaelis-Menten equation, one can estimate the concentration (or dose) of methanol, M , that puts the folate pathway into saturation, as follows:

$$1.1 = 2.0 \times \frac{M}{8.7 + M}; M = 11 \text{ mM or } 210 \text{ mg/kg, for a person with } 60\% \text{ body water}$$

The calculated value of M , though only a crude estimate, is not far from the low end of the range of methanol doses considered potentially significant clinically (0.3 g/kg). For reference to the situations of concern in this report, a 210 mg/kg body burden of methanol would be achieved if a 70 kg person breathing at 20 m^3 /day (twice resting) was exposed for an hour to roughly 18,000 mg/m^3 , absorbing 100% of the material inhaled. Since worst-case conditions for single exposures present about 200 mg/m^3 for the period spent in a hot-soak garage (15 minutes or less), the likelihood of overwhelming the folate pathway in such situations seems remote. The same conclusion also would probably apply to attendants in filling stations, who may be exposed to 50 mg/m^3 of methanol vapors for minutes at a time, many times a day.

Nevertheless, low-level exposures to methanol do cause small increases in blood and urine formate levels. Baumann and Angerer (1979) measured formate in 20 workers in a printing office who were exposed to an estimated methanol concentration of between 111 and 174 mg/m^3 of methanol throughout the work day. Over the course of a day, the blood level of formate rose an average of 4.7 mg/l (3.2 mg/l before the work

shift to 7.9 mg/l , when work ended), and urinary formate rose an average of 7.1 mg/l (13.1 to 20.2), both increases statistically significant. Every worker registered an increase for both parameters. A control group maintained relatively stable levels through the day (5.3 mg/l blood; 11.8 mg/l urine). Heinrich and Angerer (1982) performed a similar study in a chemical plant measuring methanol (blood and urine) and urinary formate in 20 workers exposed throughout the work day to 120 mg/m^3 methanol vapor, geometric mean (range of 48 to 300 mg/m^3). At day's end, blood and urine methanol in workers were 8.9 and 21.8 mg/l , respectively; a control group registered a mean blood value of less than 0.6 mg/l and a mean value in urine of 1.1 mg/l . Urinary formic acid was significantly higher in the workers (17.2 mg/l) than in the controls (12.7 mg/l).

Stegink et al (1981) assayed formate in the blood and urine of six adults who were given 200 mg/kg aspartame, equivalent to 20 mg/kg methanol. Blood methanol peaked at 26 mg/l , but no increase in blood formate was detectable over a pre-exposure value of 19.1 mg/l . Urinary formate increased in the 0-to-4 and the 4-to-8 hour post-ingestion periods to 2.9 and 2.4 times the pre-exposure levels (expressed as μg formate/ mg creatinine); after the 8 hours post-ingestion period, urinary formate was at background levels. In addition, ophthalmologic examinations conducted 24 hours post-ingestion were all normal.

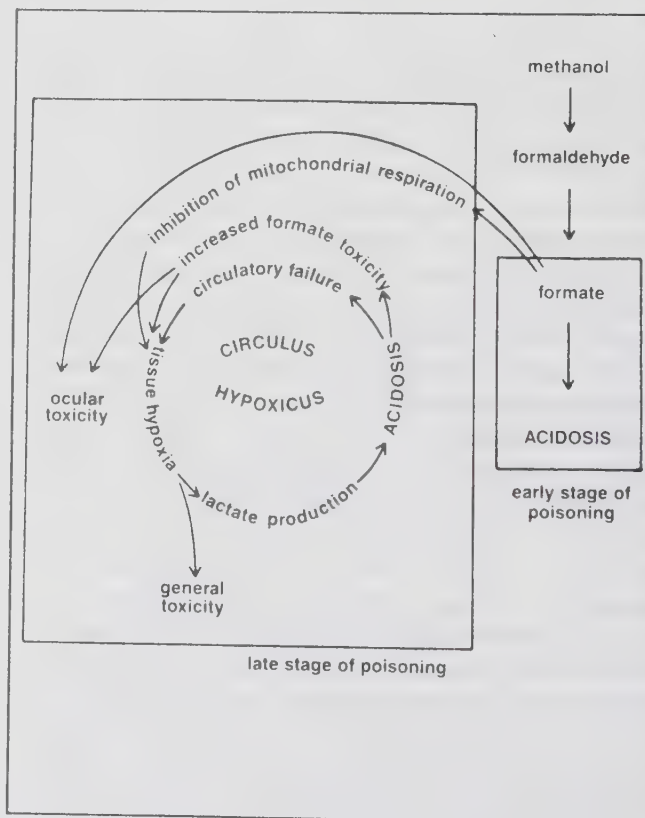


Figure 8: The circulus hypoxic: a proposed mechanism for toxicity of methanol in humans

Source: Jacobsen and McMartin, 1986
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These studies present a wide variability in the background levels of blood formate. The source of the differences has not been identified, but may relate to the methods used to assay for blood formate, and, possibly, to dietary or other lifestyle or ethnic differences between the occupationally exposed populations in West Germany (Baumann and Angerer, 1979) and the adults studied in the United States (Stegink et al, 1981). These studies demonstrate that methanol exposures that do not challenge the metabolic limits of the folate pathway, nonetheless, do generate small amounts of formate in blood and urine. In the Baumann and Angerer (1979) study, workers were exposed to about 140 mg/m³ of methanol for 8 hours; assuming 20 m³/day ventilation and 100% absorption, the dose to a 70 kg person would be 13 mg/kg. According to Sedivec et al (1981) absorption might be as low as 60%, which would lower this estimate to perhaps 8 mg/kg. These investigators noted that blood formate increased from 3.2 mg/l to 7.9 mg/l over the course of the work day. In the aspartame study of Stegink et al (1981), the dose was higher (20 mg/kg), but the high background level of blood formate (19.1 mg/l pre-exposure) may have masked any subtle increases that the aspartame may have caused. Urinary formate rose in all three studies, which indicated that, following any low-level exposures to methanol (8 to 20 mg/kg), small quantities of formate probably are generated as well; but, unless the methanol is labeled in some distinct way, incremental formate may not be discriminated from background. In worst-case single exposure conditions of concern here (see above), it is doubtful whether or not an added body burden of 1 mg/kg or less of methanol will, once metabolized, impose measurably on background levels of formate.

Although incremental formate from ambient methanol exposures may not be measurable against background levels, sufficient data on methanol and formate kinetics are available to project estimates of formate accumulation following a single brief exposure to methanol vapor. Appendix VIII presents a two-compartment model in which the entire inhaled dose of methanol enters the metabolic pathway and is converted to formate with a rate constant, k_c , reflective of blood clearance, and formate is metabolized to CO₂ with a rate constant of k_f . An example in Appendix VIII models formate accumulation in an individual whose $T_{1/2}$ for methanol clearance is 3 hours, and whose $T_{1/2}$ for formate metabolism is 45 minutes (approximately the value measured by Malorny (1969b) in humans given formate orally). For such a person, an initial body burden of 1 mg methanol/kg that results from a worst-case exposure, generates, in the model, a peak formate level of 0.0082 mM (aqueous) at 2.0 hours post-exposure (see Figure VII-1). Because background formate is about 0.2 mM, the incremental contribution from methanol exposure is about 4%. For more realistic exposures, the model predicts considerably lower levels of methanol-derived formate. Finally, the example shows the sensitivity of formate accumulation to the efficiency of formate metabolism (Table VII-1). In short, decreased efficiency raises the peak formate concentration, as expected.

Although the model is only hypothetical, it is consistent with Stegink et al's (1981) inability to detect incremental blood formate levels following a 200 mg/kg dose of aspartame (equivalent to 20 mg/kg methanol). Because 1 mg/kg methanol projects for the subject in the above example to a peak of 0.0082 mM (aqueous) formate, 20 mg/kg would project proportionally to 0.16 mM or 7.4 mg/l; in terms of blood concentration, this peak value (assuming relatively even distribution of formate in body water) corrects to 6.1 mg/l. In Stegink et al's subjects, the pre-exposure blood formate level averaged 19.1 mg/l, roughly 3 times greater, and, in addition, displayed considerable variability around the mean. Thus it is not surprising that a relatively small and transient peak of blood formate, with its own inter-individual variability, was not detected in this study.

V. EVALUATION AND RECOMMENDATIONS

DISCUSSION

Exposure to methanol vapors will increase within the general public in the event methanol becomes a widely used vehicular fuel. The principal objectives of this report are (1) to evaluate whether or not health effects may be associated with such exposures, (2) to identify areas in which our knowledge is insufficient to draw conclusions, and (3) to recommend research that will help resolve current uncertainties. Based on EPA projections, the highest exposure levels will be encountered in personal garages during engine hot-soak and in filling stations during self-service operations, and, with increasing methanol penetration into the motor vehicle fleet, in public parking garages. These situations involve single exposures of less than 15 minutes to no more than about 200 mg/m³ methanol vapor. The methanol body burden that will result from a single worst-case exposure will be less than 1 mg/kg.

Methanol has been long recognized for its acute toxicity in humans, most frequently in association with ingestion of methanol, or wood alcohol-tainted beverages. The literature is replete with case histories of methanol poisoning, and its syndrome is well-characterized. Acute effects appear after a symptomless latent period of approximately a day, and consist of an acidosis with a superimposed ocular toxicity. Methanol intoxication may lead to blindness, and is potentially lethal. Susceptibility among individuals to methanol's acute effects is highly variable, but 300 mg/kg is considered to be at the low end of the dose range considered lethal. Variability may be associated with concurrent ingestion of ethanol, which slows the progression of methanol's toxicity, or with dietary status, which may play a role in susceptibility.

The recognition in the 1950s of the non-human primate as a model of human intoxication spurred research efforts that have produced a vastly improved understanding of methanol's toxic mechanisms. Methanol's toxicity is primarily attribu-

table to its metabolite, formate, which participates in the processes that lead to acidosis and visual disturbances. Methanol itself, unless taken in narcotic doses, is not considered the toxic principal in acute poisoning. In non-human primates, ocular effects are associated with blood formate concentrations in excess of 5 to 10 mM and require a day or more to express themselves morphologically (Martin-Amat et al, 1977; Martin-Amat et al, 1978). A methodical examination of potential effects in the visual system from lower levels of formate is lacking.

Two Russian human clinical studies suggest acute (i.e., during or immediately following exposure) neurobehavioral responses from brief exposures to methanol at concentrations below 5 mg/m³ (Chao, 1959; Ubaydullayev, 1967). These responses were all sensory in nature, and involved olfaction, dark adaptation, and EEG patterns. The discussions in Section III and Appendix IV review these studies and present reasons for caution before accepting the authors' conclusions. The Russian findings must remain tentative, at best, pending replication.

The dose-effect relationships for methanol's chronic effects in humans are not well-described. On the basis of clinical case reports and a small number of epidemiologic studies, it appears that prolonged exposures to levels above the ACGIH TLV (260 mg/m³) may produce effects akin to the symptoms described for acute toxicity, but less severe. These include headache and blurred vision. Acute and chronic effects may, to some extent, share common pathways of action.

The data base for animals exposed chronically to methanol is very limited, especially in non-human primates. In one chronic feeding study, infant stump-tail macaques ingested up to 2.7 g aspartame/kg/day, i.e. 270 mg methanol/kg/day, for nine months with no physiologic or behavioral effects observed (Reynolds et al, 1984; Suomi, 1984). Histopathologic analyses were not included in these studies.

Cynomolgus monkeys exposed subchronically (6 hours per day, 5 days per week for 4 weeks) to methanol vapors at concentration levels of 650, 2,600, and 6,500 mg/m³ displayed no signs of toxicity either during the exposure period or at post-exposure necropsy (Andrews et al, 1987). The latter included histologic analyses of several tissues, including conducting airways and lung, liver, kidney, and those tissues associated with the visual system. The New Energy Development Organization (NEDO) sponsored studies, in which cynomolgus monkeys were exposed for up to 30 months to 13, 130, and 1,300 mg/m³. As discussed in Section III, the summary report issued by NEDO indicates potential chronic effects in the central nervous system at the lowest exposure level, but the details supplied in the report are insufficient to enable a critical review of the study, and further evaluation may be necessary.

Although non-primates do not serve as adequate models for the acute toxic syndrome, they may serve as subjects that can be used to explore for effects of methanol that are unrelated to its metabolism (i.e., direct effects). So far the data are limited, and do not reveal a firm indication that effects would result

from repeated human exposures under anticipated conditions. (Section III and Appendix IV)

A study of Sprague-Dawley rats (Appendix V) exposed for 4 weeks to methanol vapors (650, 2,600, and 6,500 mg/m³) reports no exposure-related effects, except for increased discharges around the nose and eyes (Andrews et al, 1987). Cynomolgus monkeys exposed to the identical regimen showed no such effects (Andrews et al, 1987). In light of the latter observation, the biological significance of the increased discharges in rats is uncertain. In a behavioral teratology study, Infurna and Weiss (1986) report decrements in suckling and homing behaviors of rat pups (Long-Evans) born to mothers that had received methanol in their drinking water for three days during the third week of pregnancy (Appendix V). The maternal dose level was 2.5 g/kg/day, a level at least three orders of magnitude higher than the doses that will occur during expected worst-case exposures. Because of the absence of data on these behavioral endpoints at dose levels relevant to expected exposure conditions, the observations of this study remain interesting, but of limited value. Finally, as part of the large-scale research program sponsored by the New Energy Development Organization (NEDO, 1986), rats and mice were subjected to long-term exposures to methanol (13, 130, and 1,300 mg/m³) to examine for toxicologic, as well as carcinogenic, effects (Section III). The summary report issued by NEDO indicates that rodents do not experience effects at 130 mg/m³. However, as mentioned in the discussion of these studies in Section III, the results are not presented with sufficient detail to allow for critical review.

As indicated, single worst-case exposures will produce methanol body burdens of less than 1 mg/kg. Specifically, a 70 kg person breathing at a rate of 20 m³/day exposed to 200 mg/m³ of methanol for 15 minutes (i.e., hot-soak, worst case exposure), absorbing 100% of the inhaled vapor, receives a dose of 0.6 mg/kg (for 60% body water, this is an aqueous concentration of 0.03 mM). More realistically, a five-minute exposure to 50 mg/m³ while refueling at the same ventilation, results in 0.05 mg/kg, or 0.003 mM. Background blood concentrations of methanol are about 0.75 mg/l, or, correcting for blood's water content (83%), about 0.9 mg/ml, or 0.03 mM aqueous concentration. Thus, for worst-case single exposures, the inhaled body burden of methanol will be equivalent to its pre-existing background level, but for more realistic exposures, it will be perhaps 10 times less. For comparison, aspartame in the diet is expected to produce additional methanol burdens that average between 0.3 and 1.1 mg methanol/kg/day (exclusive of carbonated beverages), and a single 12-ounce beverage will contribute about 20 mg, or 0.3 mg/kg, for a 70 kg person (Stegink, 1984).

The more important issue focuses on the generation of formate, and its potential toxicity. As indicated in the report, ambient exposures will not come near to challenging the folate pathway's formate metabolizing capacity, and, therefore, formate will not accumulate to frankly toxic levels. In one experiment (Stegink et al, 1981), in which adults were given

200 mg aspartame/kg orally (20 mg methanol/kg), increased formate was undetectable in blood against a background of 19.1 mg/l (0.4 mM). Urinary formate, however, increased about 2½-fold in the 8-hour post-ingestion period. In a second study (Baumann and Angerer, 1979), workers in a printing office who were exposed for the 8-hour work day to a mean 140 mg/m³ methanol vapor registered an increased blood formate of 4.7 mg/l (0.1 mM) at day's end against a pre-exposure level of 3.2 mg/l (the levels in unexposed controls was 5.3 mg/l); urinary formate increased as well. The relatively lower background in the latter study allowed the detection of the increased formate in blood. For such low-level methanol exposures, the increases of formate detectable in blood and urine reflect normally operating pathways of methanol metabolism.

As discussed in Section IV, total methanol exposure in these studies exceeds, by almost an order of magnitude, any single exposure expected from automotive or refueling vapors. The data from these studies combined with hypothetical modeling calculations suggest that single worst-case ambient exposures, i.e., 15 minutes exposure to 200 mg/m³ at twice resting ventilation, will, in a normal person, produce a temporary rise in blood formate of negligible magnitude (4%) compared to background levels. Exposures in traffic situations (Table 3), assuming 100% fleet penetration, will produce even less.

CONCLUSION

If methanol were to produce health effects in normal subjects at or near the exposure levels (time and concentration) of concern, such effects would likely not be attributable to the generation of formate. Susceptibility to methanol-derived formate remains unknown among people with dietary or metabolic deficiencies (e.g., conditions affecting the levels of liver folate or vitamin B₁₂), and remains unknown, as well, as a function of age. However, if formate is not active toxicologically and effects appear, an alternate mode of action (e.g., direct action of the alcohol) would need to be investigated.

The exposures of immediate concern will occur daily, but they will be brief and intermittent. If chronic exposures to equivalent concentrations do not produce health effects, one's confidence that anticipated conditions present no health hazard would certainly be enhanced. However, a firm conclusion regarding chronic exposure cannot yet be drawn. To this point, no human occupational studies have reported effects linked to chronic methanol exposures below the TLV (260 mg/m³). On the other hand, careful investigations of people exposed to lower levels have not been conducted, but would probably be very useful.

An analysis of the available peer-reviewed literature produces no evidence upon which to base a conclusion that exposure to low levels of methanol vapors will result in adverse health effects. This conclusion applies only to exposures that will occur as a result of methanol's normal use as a vehicular fuel, and not to exposures that may occur from ingesting methanol fuels, or from spillage.

RESEARCH OPPORTUNITIES AND ISSUES

The analysis presented in the report indicates that adverse effects are not expected as a result of exposure to methanol emissions from motor vehicles. Although several uncertainties have been identified regarding methanol's potential health effects, none are of a magnitude sufficient to justify investment of funds into a major program of research. Nevertheless, should methanol become widely implemented as a motor vehicle fuel, billions of gallons per year of this substance would be introduced into the fuel marketplace. With such large-scale usage, exposure to methanol emissions, though resulting in very low doses, is likely to be very widespread. Given this enormous scale, it is important to identify research issues and opportunities that, if addressed with focused research, would further reduce the level of uncertainty about health effects.

Much of the research to date has focused on the toxicity of methanol that follows large acute doses taken orally, and on refining clinical intervention strategies to treat that toxicity. Considerably less attention has been directed toward studying the effects that might occur following low-level chronic inhalation and the mechanisms that might be responsible. This report has reviewed the few studies concerned with low-level exposure, and has attempted to glean from the high-exposure studies the evidence that can be applied to the low-dose issues. Nonetheless, gaps in our knowledge about methanol's biological effects remain, and bridging them would provide the public with increased confidence in methanol technology.

At present, the worldwide level of effort in addressing methanol's health effects is quite small, and could be expanded to tackle specific research issues. The following discussion identifies six research opportunities that, if undertaken, would contribute valuable information on methanol's potential health effects. These research areas are not listed in priority order, and they do not represent a program that could be undertaken only with the limited resources of the Health Effects Institute as it is currently funded. Some of the problems, in a more refined form, may form the basis for future HEI work. Other problems will have to await increased interest from the relevant industrial, governmental, and scientific communities as the prospects for methanol fuels become clearer. In addition to evidence gathered from new research projects, important information may also be acquired from a re-analysis of Japan's New Energy Development Organization's study on non-human primates, and from unpublished data that may be available from studies conducted on the effects of aspartame that may be related to methanol.

The specific research opportunities are:

1. Investigate dose-effect and time-course relationships between formate levels in blood and effects to the visual system. According to current knowledge, the toxicity to the visual system in methanol poisoning is attributable to the generation and accumulation of circulating formate. However, the blood formate, in such cases,

achieves levels much higher than those that will arise from environmental exposures of the public to vehicular methanol vapors. Thus, research is suggested that will define the relationship between the concentration and duration of formate in the blood and the signs of toxicity to the visual system.

2. *Investigate local metabolism in the eye or optic nerve to determine if formaldehyde or formate is generated locally.* As indicated in the report, formaldehyde is not believed to be responsible for the known toxic effects of methanol. Methanol's metabolites, formaldehyde and formic acid, are generated mainly in the liver, and, at high enough doses, formate circulates to produce toxicity in the visual tract. One remaining uncertainty though, is whether metabolic systems are present in retinal or optic nerve tissues that also may generate toxic metabolites locally. The presence of such systems could have implications for evaluating the potential consequences of chronic, low-level exposure.
3. *Study potential susceptibility to low levels of methanol among people with dietary folate deficiency (or other suspect conditions) and within various age groups.* The report concludes, "if methanol produces health effects in normal subjects at or near the exposure levels of concern, such effects would likely not be attributable to the generation of formate." One of the major implications of this statement is that a similar definitive conclusion cannot be drawn for individuals who may be susceptible to methanol's effects. Furthermore, the Clean Air Act is written to ensure that potentially sensitive individuals remain protected. Thus, a potentially important research activity is to identify candidate determinants of susceptibility, and to develop strategies to study their role in modulating susceptibility within the population. To this end, the use of animal models may be appropriate.
4. *Conduct in-depth epidemiology study of workers exposed to methanol vapors.* As discussed in the report, studies have been conducted on various occupational cohorts exposed to methanol vapors that have measured blood and urine levels of methanol and formate. However, health-related parameters were not included in the design of these surveys. Methanol is used widely enough in various industries such that exposure information can be collected on selected cohorts and combined with data from health records, clinical examinations, and questionnaires to determine the extent to which chronic exposure to methanol affects workers, if at all.
5. *Conduct chronic animal experiments.* The most comprehensive animal research program undertaken to date was the one funded by the New Energy Development Organization. The report (Section III) indicates some of the difficulty understanding fully the findings that emerged from that program, particularly with reference to low-level chronic effects in non-human primates. A further difficulty arises with that study's design. Specifically, chronic exposures in the study occurred at methanol levels of 13, 130, and 1,300 mg/m³. Even if a no-observed-effects-level of 130 mg/m³ in this experiment (as the report suggests) is validated, uncertainty still persists about the possibility of effects occurring at levels somewhere between 130 and 1,300 mg/m³, a range that includes exposure levels that may occur in several scenarios covered in Section II. Thus, a chronic study conducted with an appropriate animal model using well-chosen exposure levels, and engaging a full quality control and peer-review process, would constitute a valuable contribution.
6. *Conduct dose-effect studies that address behavioral teratology.* As discussed in the report (Appendix V), exposure of gravid rats to a single high dose of methanol produced behavioral decrements in pups tested in the early post-natal period. Unfortunately, data are lacking that describe the relationship between the level of maternal exposure to methanol and the behavioral performance of the offspring.

APPENDIX I

EXPOSURE SCENARIOS, OFFSETS, AND SOURCES OF EXPOSURE DATA IN TABLES 3 THROUGH 5

The material in this appendix amplifies the discussions in Section II that describe the exposure levels of methanol that EPA analyses project will occur in different exposure scenarios. The following sections (1) describe the specific exposure scenarios that have been modeled, (2) discuss offsets as they relate to the data presented in Tables 3 through 5 of the report, and (3) identify the sources of data found in Tables 3 through 5.

(1) SCENARIOS

Street Canyon

Typical: four-lane canyon with a traffic load of 800 vehicles per hour; exposure on adjacent sidewalk

Severe: six-lane canyon with a traffic load of 2,400 vehicles per hour; exposure on adjacent sidewalk

Roadway Tunnel

Typical: 1,000 to 2,000 foot tunnel, with average daily traffic of between 10,000 and 15,000 vehicles per day, with longitudinal or semi-transverse ventilation at a rate of approximately 0.17 m³/sec per lane-meter; exposure in vehicle

Severe: tunnel over 5,000 feet long with average daily traffic in excess of 15,000 vehicles per day, transversely ventilated with a ventilation rate less than 0.11 m³/sec per lane-meter; exposure in vehicle

Expressway

Typical: a four-lane roadway with a traffic load 1,400 vehicles per hour and a 1 m/sec wind 315 degrees to the direction of travel; exposure in the vehicle

Severe: a ten-lane roadway with a traffic load of 3,675 vehicles per hour; same wind conditions; exposure in vehicle

Off-Road: exposure 100 meters downwind of an eight-lane highway carrying 15,000 vehicles per hour (e.g., morning rush hour); wind perpendicular to road

Parking Garage

Trip Start (Idle)

Typical: above ground naturally ventilated garage

Severe: underground garage; exposure is at lowest parking level 20 minutes after the garage is emptying from a full condition

Hot-soak

Typical: above ground, naturally ventilated garage; all vehicles in a full garage within the first hour of hot-soak

Severe: same as typical except underground

Personal Garage

Trip Start (Idle)

Typical: garage door raised; 30-second warm-up; natural ventilation

Severe: same as above with 5-minute warm-up

Hot-soak

Typical: garage door closed; ventilation rate of 20 cubic feet per minute for typical sized garage (62 m³)

Severe: door closed; no ventilation

note: The descriptions above are based on material presented by Ingalls and Garbe, 1982, and Harvey et al, 1984.

(2) OFFSETS

Automobiles are designed to limit their emissions of specific pollutants to certification standard levels. However, even with proper maintenance, emissions tend, with time, to exceed those standards. With improper maintenance, the emission levels that exceed the standards increase even more. The ratio of the actual emission level to the certified level is referred to as the offset.

For the analyses summarized in Tables 3 through 5, the offsets reflect both properly maintained and malfunctioning (i.e., improperly maintained) vehicles. For the traffic and parking garage scenarios, the offsets shown in those tables reflect average fleet-wide emission levels, taking into account the expected fraction of automobiles that are in a particular malfunction mode (see calculation below); for the personal garage scenario, the offsets for solitary vehicles are used.

Gold (1985) defines a malfunctioning and a properly functioning vehicle as follows: "Malfunction emission rates can be determined by multiplying the maximum likely emission rate under carbon-based standards by the ratio of the average of the prototype vehicle emission rate (without respect to any particular standard) to the average emission rate for properly functioning vehicles. For exhaust emissions, a malfunctioning vehicle is defined as a non-catalyst configuration and a functioning vehicle as one which meets current CO and NO_x standards. For hot-soak emissions, the ratio (i.e., offset) is based on testing...with its (the vehicle's) canister functioning and also with its canister disabled."

Gold (1985) cites other EPA references indicating that "approximately 25 percent of all LDGVs (light duty gasoline vehicles) had experienced in-use catalyst removal, air injection system tampering, or misfueling, resulting in significantly increased exhaust emissions. MOBILE3 projects that operator induced evaporative system disablement averages 2 percent. Evaporative system related failures (involuntary) are estimated at 8 percent. Thus a total of 10 percent of all evaporative systems may be considered to be operating in a malfunction mode."

The formula used to calculate the fleetwide average offsets used in Tables 3 through 5 for traffic and parking garage scenarios is adapted from Gold (1985) as follows:

$$\text{Fleetwide Offset} = (\text{Prop Frac}) \times (\text{Offset}_{\text{prop}}) + (\text{Malf Frac}) \times (\text{Offset}_{\text{malf}}),$$

where Prop Frac = the fraction of the fleet operating properly (0.75 for exhaust, 0.90 for evaporative)

Offset_{prop} = the offset for vehicles operating properly. MOBILE3 projects a value of 1.37 for exhaust emissions and 1.33 for evaporative emissions

Malf Frac = the fraction of the fleet malfunctioning (0.25 for exhaust, 0.10 for evaporative)

Offset_{malf} = the ratio of emission level from an individual malfunctioning vehicle to the level for a vehicle functioning properly. The Offset_{malf} for methanol is dependent on operating mode as follows:

Mode	Ratio
FTP	4.6
HFET	159
Hot-soak	4.8
Idle	34

(3) SOURCES OF DATA FOR TABLES 3 THROUGH 5

Street Canyon, Roadway Tunnel, Expressway (except off-road), and Parking Garage

- Data for severe conditions are those given in Gold (1985)
- Data for typical conditions are based on the severe level in Gold (1985) multiplied by the ratio of severe/typical emissions for each exposure scenario in Harvey et al (1984). For example, Harvey et al (1984) project that, for a street canyon (and all vehicles performing to certification — i.e., offset of 1.0), typical conditions require 107 g/mile, average light-duty vehicle emission of methanol vapor, to achieve a methanol concentration of 4.5 mg/m³ while severe conditions require only 16 g/mile to achieve the same ambient concentration (see Harvey et al, Table 4). Thus, given that severe street canyon conditions, with fleetwide average offsets of 1.0, produce 0.25 mg/m³ (Table 3, street canyon), then typical conditions are expected to produce (16/107) x 0.25 mg/m³ or 0.04 mg/m³, as shown in Table 3.

Off-Road Expressway

The off-road values in Table 3 are obtained by applying the given likely certification level (0.023 g/mile) to data in Harvey et al (1984), Table 4. In the latter reference, the authors project that emission levels of 35.7 g/mile, with an offset of 1.0, will achieve an off-road methanol level of 4.5 mg/m³. To calculate the off-road level for Table 3, one needs to further consider that Harvey et al equate light-duty hydrocarbon emissions to 0.82 of the fleet average, while Gold (1985) assigns a value of 0.62. Since all other values in Tables 3 through 5 are based on Gold (1985), one needs to factor in a correction of 0.82/0.62 to calculate off-road expressway levels that are consistent with the other data shown in the tables. Thus, for an offset of 1.0, the Expressway Off-road Level =

$$(0.023/35.7) \times 4.5 \times (0.82/0.62) = 0.004 \text{ mg/m}^3$$

Personal Garage

All data from Gold (1985)

APPENDIX II

HUMAN STUDIES: REPEATED OR PROLONGED EXPOSURES

Reports of effects from chronic or repeated methanol exposures have appeared infrequently in comparison to reports of acute toxicity. Although details of exposure (duration, concentration) are usually missing, the effects of prolonged exposure are qualitatively very similar to those reported for acute cases, consisting of central nervous and visual disorders. The studies described are divided into case reports and epidemiologic studies.

CASE STUDIES

The first of these was a 1901 report (De Schweinitz, 1901) of a man who became blind after periodic exposure to varnish dissolved in methanol, and the use of methanol to clean his face and arms over a period of three years (also reported in Wood and Buller, case A-25). The Wood and Buller series of case reports in 1904 included several cases that indicated methanol toxicity from extended exposure (Wood and Buller, 1904; Buller and Wood, 1904): failed vision, headache, and vomiting in a man who dyed and cleaned clothes in alcoholic preparations; length of exposure unspecified (Case B-66); deteriorating vision in a man who frequently drank Jamaica ginger (pure methanol) as an alcoholic substitute; an acute dose took his life (Case B-86); a woman who daily took three or four tablespoonsful of Jamaica ginger went practically blind (Case B-89); a woman who, for weeks, used wood alcohol to heat her rheumatic bath and as a cleansing application to face and head presented with impaired vision and partial pallor of the optic nerve (Case C-2); visual loss occurred in a woman who, for two or three months, had used an alcohol-fueled lamp to heat water in a poorly ventilated space; she recovered after her doctor told her to cease exposure (Case C-4); a man who, varnished beer vats for a living, used shellac cut with methanol, and experienced "constitutional symptoms" and "foggy vision" from inhaling the vapors; length of exposure unspecified (Case C-6); and a workman in a cabinet department who cleaned his hands in Columbian spirits to remove shellac suffered from impaired vision (Case C-9).

Severe visual effects were subsequently reported in men exposed to methanol vapor when methanol was used as a paint remover and for mixing shellac (Hawes, 1905); or for varnishing beer vats for periods of 3 to 5 days (Tyson, 1912; Wood, 1912). In each of these instances CNS symptoms (headache, dizziness, nausea, numbness) preceded or accompanied the development of visual symptoms. In a 1905 report (Jelliffe, 1905), CNS symptoms were described in two men who inhaled fumes from shellac dissolved in methanol, but no visual sequelae followed. In none of these reports was the actual level of methanol exposure determined or estimated.

Ziegler (1921) described a man who visited a china cement factory for one hour each day. Methanol was found to be a constituent of the cement. The man had experienced, for several months, failing vision and contraction of visual fields. When visits to the cement factory were stopped, the man slowly recovered and then maintained normal vision. Ziegler (1921) also described a painter who inhaled fumes while varnishing an engine room in a submarine for three days. He was "dizzy" the first day, "hilarious" the second, and "nervous" the third. He also suffered gastric pain and insomnia, soon followed by ptosis (drooping of the eyelid), and blindness. Ziegler also stated that these symptoms were associated with acidosis, although no clinical data were provided. As in the earlier cases, the actual level of exposure was not known.

Humperdink, in 1941, reported the occurrence of mild methanol intoxication with temporary blindness in one laborer employed in a nitrocellulose plant. This worker could presumably have experienced repeated exposures of 1,600 to 10,900 mg/m³ methanol, which was the amount measured in the air above the weighing station where the worker was employed. The authors noted, however, that over a ten-year period, no other workers had reported any symptoms of methanol toxicity. Burk (1957) also described a case of occupational poisoning attributable to methanol vapor inhalation. This worker had been employed in the methyl alcohol department of a chemical pharmaceutical factory for four years, and had previously complained of visual disorders and asthenia (weakness) of the hands and arms. Upon a two-day exposure to methanol fumes while cleaning a boiler in which crude nicotinic acid was boiled with methanol, he experienced vertigo, nausea, and visual disorders. No information on the airborne concentration of methanol in the air inhaled was provided. Ophthalmoscopic examination showed edema of the optic disc of both eyes. After five weeks, full visual acuity returned.

EPIDEMIOLOGY

A small number of epidemiology studies have been published on methanol exposure, but are generally documented inadequately. The earliest study involves 25 to 30 women who polished wooden lead pencils with varnish made from methanol; many washed their hands in the alcohol to remove the shellac (Tyson, 1912). All of the women reported headaches, and some had gastric disorders during working hours; two reported visual disturbances. They frequently took breaks to get fresh air. The airborne concentration of methanol was unknown. Greenburg et al (1938) studied 19 workers employed in the manufacture of "fused collars." These workers used solutions of three parts acetone to one part methanol to impregnate collars, which were then steam pressed. Concentrations of acetone and methanol in the work room were

measured to be 96 to 108 mg/m³ and 29 to 33 mg/m³, respectively, and a "strong odor" of solvent was perceptible. The shortest period that any of these workers had spent fusing collars was nine months, and the longest was two years. No central nervous system or visual anomalies in any of these workers were reported.

In 1955, Kingsley and Hirsch reported frequent and persistent headaches in clerical workers located close to spirit duplicating equipment that used methanol-based duplicating fluid. The most severe headaches were reported to occur in personnel who actually operated the equipment. The onset of symptoms coincided with the beginning of cooler weather, which required the closing of windows and doors, thus inhibiting ventilation. No visual effects or other permanent sequelae were reported. Kingsley and Hirsch (1955) measured methanol concentrations as high as 490 mg/m³ in the air surrounding the duplicating equipment after 60 minutes of operation, and approximately 130 mg/m³ about ten feet away from the device. The methanol concentration around the device in question always exceeded 260 mg/m³. No information on the number of employees exposed or affected, or on the actual duration of methanol exposure, was provided in this report.

More recently, the National Institute for Occupational Safety and Health (NIOSH, 1981) reported that 45 percent of "spirit" duplicating machine operators at the University of Washington experienced "some" symptoms (blurred vision, headache, nausea, dizziness, and eye irritation) consistent with the toxic effects of methanol. Apparently, no information on the actual level of duration of methanol exposure among these employees was collected. When NIOSH measured airborne methanol concentration for 25 minutes in the vicinity of the duplicators when windows and doors were open, the average was 1,330 mg/m³.

In 1984, Frederick et al of NIOSH published a study of teacher aides who worked at or near spirit duplicators that used a 99% methanol duplicator fluid. The exposures ranged from 1 hour per day for 1 day per week to 8 hours per day for 5 days per week, and had been occurring presumably for about three years. Ventilation was either inadequate or totally lacking. Since the introduction of the equipment, the aides began to experience headaches, dizziness, and eye irritation while operating the machines or working near them. Fifteen-minute breathing zone samples near 21 operating machines contained between 475 and 4,000 mg/m³ ($1,380 \pm 745$ (SD) mg/m³) of methanol vapor; 15 of these samples exceeded NIOSH's recommended 15-minute standard of 1,050 mg/m³ (or 800 ppm). The existing ventilation, when operating, lowered the levels around selected machines by an average of 58% (range: 7 to 89%); even so, methanol concentrations exceeded the 260 mg/m³ 8-hr standard for at least half of the duplicators tested. When NIOSH-fabricated enclosures were added, the unventilated values dropped by an average 96% (range: 90 to 99%). The aides also were exposed while collating and stapling papers impregnated with the fluid up to three hours earlier, and these exposures ranged from 235 to 1,140 mg/m³.

A health questionnaire survey was conducted among 84 teacher aides and 302 teachers, who served as a comparison group. Teachers, though working in the same school, probably spend significantly less time near the duplicators (and less time collating) than the aides. All aides and teachers surveyed were female. Sixty-six (79%) of the aides responded (mean age 39.8); their responses were compared to those of 66 randomly selected teachers (mean age 37.5). The respondents provided data on the prevalence of 22 specific symptoms that they experienced in the month preceding the survey. The list included symptoms considered both related and unrelated to methanol's effects. Four of the 22 symptoms were significantly higher in the aides, and all have been associated with methanol toxicity: headache, dizzy/lightheaded, blurred vision, and nausea/upset stomach (Table I-1). No other symptoms registered significant differences between the two groups, although positive trends appeared evident for burning/itching/tearing of the eyes (17 of 66 aides versus 8 of 66 teachers) and skin problems (7 versus 1, respectively).

Criteria were established to define a positive case of methanol toxicity (Table I-2). With these criteria, 30 aides and 16 teachers qualified, a difference that is significant ($p < .025$). Finally, the investigators determined that the case attack rate increased, for both aides and teachers, as a function of percent of time spent at the duplicator each week.

This study clearly stands apart from all others available because it provides data on ambient concentrations, duration of exposure, health status, and the relation between case attack rate and work-time exposed. The results suggest that chronic effects may occur when methanol concentrations exceed the TLV of 260 mg/m³. The effects in this study are similar in nature but appear less severe than those from acute intoxication.

Though of value, this study, nevertheless, has several features that prevent one from drawing a definitive causal association between methanol exposures and the effects reported. First, the study was conducted in response to the teacher aides' complaints, and thus the questionnaire data may have contained responder bias. Second, the data were based on symptom reporting only, whereas additional clinical investigations may have helped to further define the effects. Finally, information is not presented to exclude the possibility that the symptoms might have arisen from other chemicals or solvents that may have been in the teacher aides' environment. Despite these shortcomings, the study by Frederick et al is of relevance.

Other studies have measured methanol and formate in the blood and urine of workers exposed during the 8-hour day to between 100 and 200 mg/m³ of methanol vapors (Baumann and Angerer, 1979; Heinrich and Angerer, 1982). Although these studies were predicated on issues of occupational health related to methanol exposure, no health data are provided. In none of these studies do the investigators imply that the workers studied had suffered health effects.

Table II-1 Symptoms Significantly ($p < .05$) More Prevalent in Teacher Aides than Teachers

Symptom	# Teachers (n = 66)	# Aides (n = 66)	Ratio
Headache	12	23	2
Dizzy/lightheaded	1	20	20
Blurred vision	1	15	15
Nausea/upset stomach	4	12	3

Adapted from: Frederick et al, 1984

Table II-2 Criteria for Defining Methanol Toxicity

1. Visual changes or blurred vision
2. One acute symptom (headache, dizziness, numbness, giddiness, nausea, or vomiting) and one chronic symptom (usually tired, muscle weakness, trouble sleeping, irritability or poor memory)
3. Two acute symptoms
4. Three chronic symptoms

Adapted from: Frederick et al, 1984

APPENDIX III

ACGIH TLV FOR METHANOL

Reprinted, by permission, from: American Conference of Governmental Industrial Hygienists, Inc. (ACGIH). Documentation of the Threshold Limit Values and Biological Exposure Indices. Fifth edition. Cincinnati, OH, 1985.

METHYL ALCOHOL

CAS: 67-56-1

Methanol

CH₃H

Skin

TLV-TWA, 200 ppm (≈ 260 mg/m³)

TLV-STEL, 250 ppm (≈ 310 mg/m³)

Methyl alcohol is a mobile, highly polar, flammable liquid. Its physicochemical properties include:

Molecular weight: 32.04

Specific gravity: 0.7915 at 20°C

Melting point: -97.8°C

Boiling point: 64.5°C

Vapor pressure: 92 torr at 20°C

Vapor density: 1.11 (air = 1)

Closed cup flash point: 54°F (12°C)

Autoignition temperature: 878°F (470°C)

Explosive limits: 6.7% and 36.5% by volume in air

It is miscible with water, ethyl alcohol, ether, and many other organic solvents.

Methanol is used as a solvent for nitrocellulose, ethyl cellulose, and various natural and synthetic resins; as a denaturant for ethyl alcohol; as an antifreeze; and in the manufacture of formaldehyde and many other chemicals, notably methyl derivatives.

According to Henderson and Haggard,¹ methanol is slowly eliminated from the body, hence repeated exposures result in an increasing concentration in blood and tissue. McNally² stated that occupational methanol poisoning has frequently caused death or blindness. Several cases resulted from work in confined spaces, e.g., varnishing beer vats. Headaches and blurred vision were reportedly frequent symptoms. He believed that 8 grams would seriously affect the eyes. (Such a dose could result from inhalation of 800 to 1000 ppm for eight hours.) According to McNally, workroom concentration of 500 to 6000 were found; he recommended that levels be kept below 1 ppm.

Browning³ stated that cases of chronic poisoning from repeated exposure to methyl alcohol vapor were manifested by conjunctivitis, headache, giddiness, insomnia, gastric disturbances and failure of vision. In one fatal case of occupational methanol intoxication by inhalation, a female worker was exposed about 12 hours.⁴ A post-event study of the process revealed methanol vapor concentrations ranging from 4000 to 13,000 ppm. Henson,⁵ in a review of methanol toxicity, mentioned a report of chronic poisoning, with marked diminution of vision, resulting apparently from exposure at 1200 to 8000 ppm for four years. Other workers in the area were not affected. Henson also recorded reports of headaches among workers exposed at 300 ppm during the operation of duplicating machines.

Most of the serious cases of methanol poisoning which have been reported during the last 40 years, many of them fatal, others involv-

ing permanent or temporary loss of vision, resulted from the ingestion of methyl alcohol in the belief that it was ethyl alcohol.

A study of the wood heel industry in Massachusetts⁶ showed average methanol vapor concentrations ranging from 160 to 780 ppm, with no definite evidence of injury to the exposed workers. McAllister⁷ reported concentrations between 400 and 1000 ppm in spirit duplicating processes. No mention was made of symptoms or complaints, but these concentrations were considered excessive.

Sayers and co-workers⁸ observed no symptoms in dogs exposed daily (seven days a week) for 379 days at concentrations between 450 and 500 ppm. Leaf and Zatman,⁹ after studying the rates of absorption and excretion of methanol, concluded that at 3000 ppm accumulation in the body would occur, and that the maximum safe concentration for occupational exposure was 300 ppm.

Cook,¹⁰ on the basis of the Sayers study,⁸ recommended a limit of 200 ppm. It is probable that this value incorporates a fairly large margin of safety against serious toxic effects.

According to the NIOSH criteria document for methyl alcohol, the signs and symptoms most characteristic of methanol poisoning are various visual disturbances and metabolic acidosis.¹¹ The NIOSH review of the literature failed to reveal any epidemiologic surveys sufficiently comprehensive to bear significantly on the workplace environmental limit. A report by Kingsley and Hirsch¹² indicated severe recurrent headaches in workers exposed to methyl alcohol in concentrations between 200 and 375 ppm. Diminution of vision was reported from airborne methyl alcohol concentrations of 1200 to 8300 ppm.¹³

NIOSH therefore recommended a TWA standard of 200 ppm for methyl alcohol, the same as the time-weighted average TLV of long standing. A fifteen minute ceiling of 800 ppm, well above the TLV-STEL of 250 ppm, was recommended.¹¹

Other recommendations: ANSI (1944) 200 ppm; Sweden (1975) and West Germany (1974) 200 ppm; Czechoslovakia (1969) and East Germany (1973) 75 ppm; USSR (1972) 4 ppm.

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APPENDIX IV

DISCUSSION OF RUSSIAN HUMAN CLINICAL STUDIES

Russian investigators published papers in 1959 and 1967 that claimed neurobehavioral effects in humans who were exposed to very low exposure levels of methanol vapors (less than 12 mg/m³). In the first paper, Chao (1959) measured the threshold of olfactory perception and dark adaptation (or light sensitivity). The second, by Ubaydullayev (1967), included both of these measures in addition to the EEG conditioned reflex threshold. These studies are presented and analyzed in detail in the discussion that follows.

OLFACTORY THRESHOLD

Of the three parameters tested in the two studies, olfactory threshold is the only one that other investigators also have tested. These other studies, two in all, report olfactory thresholds between two and three orders of magnitude higher than Chao and Ubaydullayev. Scherberger et al (1958) studied thresholds in three subjects and reported a minimum identifiable concentration of 1,950 mg/m³. May (1966) studied 16 subjects and reported a minimally perceptible level of 7,670 mg/m³.

Several problems, related to the procedural aspects of olfactory testing, call into question the results of the two latter studies. Basically, the sensory threshold is the level of exposure that is detected accurately 50 percent of the time. Threshold determination depends on the systematic acquisition of subject-specific curves that plot response as a function of exposure concentration.

Another important consideration in odor-threshold detection is the nature of olfactory physiology. The presentation of olfactory stimuli must be carefully controlled in order to assess accurately whether or not the threshold actually has been reached. With closely repeated or continuous exposures to an olfactory stimulus, the ability to detect it rapidly decreases, a phenomenon known as olfactory fatigue. When olfactory fatigue is present, progressively higher concentrations must be presented in order for the subject to detect the chemical. Naturally, this would prevent the accurate detection of a minimum threshold. Neither the Scherberger nor the May studies employed adequate paradigms for threshold detection or protected against olfactory fatigue. In the Scherberger study, no experimental details were provided, including whether several exposure levels were tested for each subject or whether subjects were exposed to high concentrations immediately prior to lower ones; also, subjects may have experienced olfactory fatigue. Likewise, the threshold in the May study should be viewed as unreliable because there is a strong likelihood that olfactory fatigue affected the results.

In contrast to these reports, both the Chao (1959) and Ubaydullayev (1967) studies apparently used more appropriate

methodology for the detection of odor thresholds. According to a comprehensive review of behavioral toxicology paradigms used in the USSR (NIOSH 1976b), Russian investigators use paradigms prescribed by a committee affiliated with the USSR Academy of Sciences. Therefore, although methodology in Russian research papers, such as the ones cited above, is often poorly explained, the procedures used are standardized, and reference can be made to the author of the method or to key papers. Standard protocols for a variety of tests, including olfactory thresholds, have been described by NIOSH (NIOSH 1976b). Those descriptions have been relied upon here to interpret the English translations of the Chao and Ubaydullayev studies.

In both Russian olfactory studies, two sniffing cylinders, one that contained pure air, and the other that contained either a methanol-air mixture or pure air, were given to subjects who were asked to sniff freely from each until ready to indicate which contained the methanol mixture. Each concentration was presented at least three times. The minimum perceptible concentration for each subject was taken as the lowest concentration detected in at least two out of three trials. Presumably, the criterion for the maximum imperceptible concentration also was correct detection on at least two out of three trials, although that is not clearly delineated in the standard protocols. Both a minimum perceptible and a maximum imperceptible methanol concentration were reported.

Using this paradigm, in the Chao (1959) study the minimum detectable airborne concentration of methanol was found to range from 4.3 to 11.1 mg/m³ and the maximum imperceptible airborne concentration was found to range from 3.7 to 10.5 mg/m³. In the Ubaydullayev (1967) study, the maximum imperceptible methanol concentration ranged from 3.9 to 9.7 mg/m³ and the minimum perceptible concentration ranged from 4.5 to 10.3 mg/m³.

Although the Russian studies apparently adhered to an appropriate methodology, the results should be viewed as provisional. An important substantive concern that forces this conclusion is the investigators' failure to document the purity grade of the methanol used. Impurities can have a marked effect on olfaction, producing false-positive results at methanol levels that are actually sub-threshold. Despite the shortcomings of the Scherberger and May studies, the large discrepancy of the results from these and the Russian set is difficult to reconcile. The possibility that impurities may have skewed the Russian results cannot be ruled out. This opinion also was expressed in the 1976 NIOSH document on occupational exposure to methanol (NIOSH, 1976a). One further issue that applies to the olfactory experiments, as well as to the dark adaptation and EEG tests, is the failure of either Chao or Ubaydullayev to describe the manner in which methanol vapor concentrations were measured. Instrumentation,

measurement technique, and calibration procedures each can serve as source of error, and distort test results. Regardless of the value of the "true" threshold, olfaction is an indication of sensory stimulation, not a toxic response.

DARK ADAPTATION

In simple terms, a dark adaptation curve describes the threshold level of light one perceives as a function of time in the dark. Normally, the threshold decreases with time (i.e., sensitivity to light increases) as adaptation continues and ultimately levels to a final value (Guyton, 1981). Cones (receptors specialized to discriminate color) and rods (do not discriminate color, specialized for dark vision) typically adapt differently; rods adapting slow over the course of 30 to 60 minutes, and cones adapt within 10 minutes, but to a much lower sensitivity than the rods. Thus, depending on test conditions, adaptation curves may demonstrate a "rod-cone break." For example, if the test spot of light is directed to the retinal periphery, which is relatively cone-poor, then the break will not occur; similarly, if the pre-adapting stimulus (the lighting condition prior to dark adaptation) is itself dim, cone adaptation already may have concluded prior to the adaptation test, and again no break will appear. The eye's sensitivity to light can change by a factor of between a half-million and a million, that is, between 5.7 and 6.0 \log_{10} units. Of these, photochemical receptivity of rods and cones account for about 4.4 ($\times 25,000$) and pupillary adjustments for about 1.5 ($\times 30$); an additional fraction of a log unit is associated with intermediate neuronal levels in the retina. Tests of dark adaptation typically probe for photoreceptivity in the retina, and uncontrolled changes in pupillary size. Extraneous factors that are distracting to the subject can confound a test's results.

Dark adaptation is inherently difficult to measure for two reasons. First, it is continually changing, so the measurement must be of short duration. Second, one uses light presentation to measure dark adaptation, and if the test light is too bright it will alter the course of adaptation. To mitigate these problems, the Russian investigators chose a von Bekesy tracking technique. For each measurement, the intensity of the test flash is increased until the subject signals that it is seen. The light stimulus then decreases in intensity until the subject signals that it is no longer visible. The light threshold is taken as the mean of these two inflections.

In both Russian studies, the subjects were exposed to methanol from the 15th to the 20th minute of dark adaptation. Dark adaptation curves are usually dynamic during this time frame and, therefore, are maximally sensitive to acute effects. Chronic conditions, such as vitamin A deficiency, manifest over the entire time course of dark adaptation, or in the final level of sensitivity.

As in the olfaction experiments, the dark adaptation studies probably were performed with standardized procedures (NIOSH, 1976). Ubaydullayev reports using an ADM adaptometer, which is one of two adaptometer recommended for use

in the Soviet Union (Chao's equipment was unspecified). In the procedure, the stimulus, transilluminated with an incandescent lamp, locates binocularly 12 degrees to the right of fixation (i.e. 12 degrees from the fovea, the point of sharpest vision in the retina). This type of stimulation usually produces a curve with some evidence of rod and cone components, with the transition occurring late in the first ten minutes of adaptation (as mentioned before, the rod-cone break will not appear with a sufficiently dim pre-adapting stimulus).

The Chao study and Ubaydullayev study each tested dark adaptation in three subjects. The subjects in the Ubaydullayev study were between 18 and 25 years old; no characteristics of Chao's subjects were given. In Chao's experiments, adaptation for each subject was measured at five different methanol concentrations (0, 1.8 to 2.4, 3.3 to 3.7, 4.3 to 4.7, and 5.7 to 6.5 mg/m^3); according to the translation, "59 determinations were done in all" and "each concentration was examined several times." In Ubaydullayev's study, four concentrations were used (0, 3.06, 3.53, and 4.11 mg/m^3), and "tests were made daily on each individual under identical conditions and at a standardized time of day." Further, "the physiological background — i.e. the normal curve of eye adaptation to the dark or sensitivity to light — was determined on 8 successive days by 15- and 20-minute pure air inhalations." These are the only details provided.

Important information that neither investigator provided concerns the number of curves run for each individual for each concentration (or how they varied) and the time points along each curve at which light detection was measured; nor did they discuss the pre-adapting stimulus, the size of the stimulus on the retina, or how they controlled fixation, all of which may influence the rod-cone break.

Both investigators reported seemingly consistent results. Chao claimed an effect at 3.3 mg/m^3 , but none at 2.4 mg/m^3 , and Ubaydullayev claimed an effect at 3.53 mg/m^3 , and none at 3.06 mg/m^3 . Actual data, however, are shown for only a single subject in each study. Furthermore, the time-course of adaptation for the curves shown in both studies is quite unusual for this kind of test, displaying an upwardly concave shape in the first few minutes. Generally, dark adaptation proceeds rapidly in the first few minutes and the shape has a downward concavity.

The few data shown from the two studies are inconsistent. In Ubaydullayev's study, 3.53 mg/m^3 produced an immediate rise in sensitivity (methanol is presented at the 15th minute of adaptation) that peaked at 20 minutes of adaptation (when methanol exposure ceased) and returned to control values at 25 minutes; 4.11 mg/m^3 produced an immediate depression that lasted until 30 minutes. The test terminated at 40 minutes. Chao's results showed an opposite trend; 3.3 to 3.7 mg/m^3 produced a subtle drop in sensitivity from the time that methanol was introduced to the end of the test (60 minutes), whereas 4.3 to 4.7 mg/m^3 produced a small initial rise in sensitivity followed by a dip below control about 20 minutes later, which persisted to the end. At 5.7 to 6.5 mg/m^3 , Chao's data

accentuated the effects reported at 4.3 to 4.7 mg/m³. If methanol produced a distinct acute effect at these concentrations, one would expect a consistent set of data from "replicate" experiments. The results presented offer no such possibility.

Several other factors are germane to this discussion. First, though olfactory sensations do not directly influence the photoreceptivity of rods and cones, they may distract a subject to the extent that his or her perception of light is mildly altered. Since the thresholds for olfaction of the methanol (and/or its impurities) overlap the levels that putatively affect dark adaptation, the possibility of distraction cannot be dismissed. Second, the reader should bear in mind that if methanol is affecting visual sensitivity in these experiments, the effect bears no correspondence to the classic visual toxicity seen after a one day latency. Post-latency toxicity results from a methanol metabolite, probably formate, whereas, in the kinds of experiments described here, the alcohol itself would be responsible. Finally, the amount of methanol absorbed into the body during the five-minute exposure is insignificant compared to background levels in the body. To illustrate, a "typical" 70 kg man with 45 liters of body water (methanol distributes uniformly to body water) breathing and totally absorbing 10 mg/m³ methanol for five minutes, would raise his concentration of methanol by 0.008 mg methanol/liter body water. In contrast, background blood levels of methanol are around 0.75 mg/l or more than 100 times greater than the amount contributed during the Russian dark adaptation, which were all run below 12 mg/m³. How this minor amount may directly affect central nervous function must remain open to conjecture.

To conclude, altered dark adaptation may constitute an "adverse effect." The apparent similarity of results from the two Russian studies published eight years apart by different investigators is noteworthy. However, if the protocols, reagents, and instrumentation remained standardized in the period that covered both studies, then one could expect similar, but not necessarily valid, results.

EEG-CONDITIONED REFLEX THRESHOLD DETERMINATION

Ubaydullayev (1967) also examined conditioned alpha rhythm amplitude using an electroencephalogram (EEG) in six subjects exposed to methanol. The methods were poorly described in the paper and, consequently, the following comments rely, again, on the NIOSH (1976b) review of Russian neurobehavioral toxicology methods.

According to NIOSH (1976b), a depression in cortical alpha wave activity is used as a conditioned response in these experiments. Various concentrations of a volatile chemical (methanol) are presented as conditioned stimuli, and the onset of a light is presented as the unconditioned stimulus. In an experimental conditioning paradigm such as this, the unconditioned stimulus (light) is one that normally elicits the conditioned response (depressed alpha wave activity). When both the unconditioned and conditioned stimulus (light and methanol, respectively) are presented together often enough, the conditioned stimulus alone (methanol) develops the ability to elicit the response. Thus, the fact that a stimulus, such as a certain methanol concentration, can be conditioned, is evidence that it has been detected. The lowest concentration of a chemical that can be conditioned is reported as the EEG-conditioned reflex threshold.

In the Ubaydullayev (1967) study, conditioning was attempted in the six subjects most sensitive to olfactory stimulation. They were exposed to methanol concentrations of 1.01, 1.17, and 1.46 mg/m³. The lowest concentration of methanol that successfully conditioned was 1.17 mg/m³, eliciting the response in two subjects. All six subjects exhibited depressed alpha wave amplitude, displaying the conditioned response when presented with 1.46 mg/m³ methanol.

Detection, as reflected by altered alpha rhythms, occurred below threshold levels for olfaction, a finding that is not uncommon. Such a response is indicative of an organism's ability to detect the presence of a substance, and provides evidence of a normally functioning nervous system.

Several of the concerns voiced earlier linger. Details about the purity grade of methanol, the subjects, the exposure protocol, and test data are lacking. At the levels used, the accumulation of methanol in the subjects is, in all likelihood, negligible compared to the normal methanol body burden. Finally, nowhere in any of these studies do the investigators guarantee that both they and the subjects remained blind to the exposure condition.

APPENDIX V

STUDIES OF REPEATED OR PROLONGED METHANOL EXPOSURE IN NON-PRIMATES

RODENTS:

Because of the superiority of non-human primates as experimental models of human methanol toxicity, few experiments on the biologic effects of methanol have been conducted using rodents in the past 30 years. In a study by Skirko et al (1976) (a Russian study cited in Rowe and McCollister, 1982), rats received oral doses of 10, 100, or 500 mg/kg/day for one month and were reported to show liver changes characterized by focal proteinic degeneration of hepatocytic cytoplasm, changes in the activity of some microsomal enzymes, and enlarged hepatic cells. [In another Russian study, rabbits exposed to 61 mg/m³ methanol for six months (duration of exposure per day not given) were reported to have ultrastructural changes in the photoreceptor cells and Muller fibers (Vendilo et al, 1971, a study cited in Rowe and McCollister, 1982).] The reliability of either of these reports remains to be established through critical review of translated articles. In an English article, White et al (1983) reported that exposure of rats (Sprague-Dawley) to airborne methanol concentrations of 260, 2,600, or 13,000 mg/m³ for as long as six weeks caused no signs of lung inflammation or irritation. Histologic analyses of lung tissue were not conducted.

In a subchronic inhalation study, Sprague-Dawley rats were exposed for 4 weeks (6 hours per day, 5 days per week) to 650, 2,600 and 6,500 mg/m³ of methanol vapor (Andrews et al, 1987). The animals were observed twice daily for signs of toxicity, and were given detailed physical examinations each week, and ophthalmoscopic examinations at pre-test and at termination. After sacrifice, the animals' organs were examined and weighed, and selected tissues from all animals in the control and high-exposure groups were examined microscopically. These included nasal turbinates, trachea, lungs, trachea, esophagus, liver, and the eye and optic nerve. The investigators report no effects, except for increased discharges about the eyes and nose. The only dose-related effect observed was mucoid nasal discharge, which the investigators believe is reflective of upper respiratory tract irritation. Though stating that this effect was dose-related, Andrews et al provide no dose-effect data on this finding. No other treatment-related effects were observed in this study.

Behavioral toxicity associated with exposure to low concentrations of methanol in laboratory rats was reported in two studies in the Russian literature (Chao, 1959; Ubaydullayev, 1967). Chao (1959) exposed groups of ten rats (of unspecified sex and strain) to methanol vapor at concentrations of 0, 1.77, and 49.77 mg/m³ for 12 hours per day ("excluding days off") for 3 months. The relationship of flexor to extensor chronaxy

was measured at unspecified intervals. According to a review of behavioral toxicology paradigms used in the USSR (NIOSH 1976b), "chronaxy is the minimum time necessary for a stimulus of twice the absolute threshold intensity to evoke a response" and is measured as muscle contractions in response to an electric current applied to the animal's hind leg. Normally, the flexor chronaxy is shorter than the extensor chronaxy, and their ratio is stated to be a relatively stable one. According to the NIOSH review, certain toxic agents have been found to reverse this relationship (NIOSH 1976b). Although the chronaximetry method as used by Chao (1959) and Ubaydullayev (1967, see below) is poorly described in both published studies, the methodological details provided are consistent with the standardized methodology summarized by NIOSH (1976b).

Chao (1959) reported that the average chronaxy ratio for rats in the high dose group significantly differed from that in the controls at week eight of exposure. The average chronaxy ratio was stated to have returned to normal during the recovery period. Effects in the low-dose group were reported to be insignificant. Although the investigators reported that dose groups consisted of 10 animals per group, they did not indicate the number of animals tested per testing interval, or the frequency of measurement of chronaxy ratios. Data were presented only graphically and actual chronaxy ratios and results of statistical analyses were not provided.

Chao (1959) also reported certain histopathological changes in the high-dose group but not in the low-dose group. The lesions included "poorly defined changes in the mucous membranes of the trachea and bronchi," hyperplasia of the submucosa of the trachea, slight lymphoid infiltration, swelling and hypertrophy of the muscle layer of pulmonary arteries, slight degenerative changes to the liver, and changes in the neurons of the cerebral cortex. A list of tissues examined histopathologically, the number of animals per group subject to pathological examination, and the incidence of tissue lesions were not provided.

Ubaydullayev (1967) exposed groups of 15 male rats (strain not specified) to methanol at average air concentrations of 0, 0.57, and 5.31 mg/m³ for 24 hours per day for 90 days. Motor chronaxy ratios were measured at 10-day intervals in five rats per group. Ubaydullayev reported that the high-dose group "manifested statistically reliable changes" in the motor chronaxy ratio beginning at week 6, and that the ratio returned to normal by the end of the recovery period (length of recovery period is not specified). Average values for the chronaxy ratios for the three groups over the study were presented graphically, but the actual data and statistical analyses were not provided.

Urinary coproporphyrin levels, whole blood cholinesterase activity, and levels of total protein and protein fractions in blood serum also were measured in 5 rats of each group (Ubay-

dullayev 1967). In the high-dose group, the investigator reported a decrease in urinary coproporphyrin levels and cholinesterase activity. Blood serum albumin levels dropped and levels of beta- and gamma-globulins increased in the high-dose group compared to the controls. These parameters were reported to have returned to normal after cessation of exposure. No effects were observed in the low-dose group. The investigator presented average values for the control, low-dose, and high-dose, but did not provide any statistical analysis of the data.

The results reported by Chao (1959) and Ubaydullayev (1967) do not provide adequate evidence of an association between neurobehavioral effects and low-level exposure to methanol in laboratory animals. Both studies are limited by the use of small numbers of animals per dose group, as well as insufficient reporting of experimental methods, study results, and statistical analyses. Furthermore, the biological significance of changes in the chronaxy ratio is uncertain. Although measurement of the chronaxy ratio in rats appears to be a standard protocol for assessing neurobehavioral toxicity in Russian research, it is not a toxicological measure reported in the U.S. literature.

DOGS:

Sayers et al (1944) exposed two dogs to about 13,000 mg/m³ methanol for about three minutes at hourly intervals eight times daily for 100 days, a total of 800 brief exposures. Both dogs were reported to have survived the exposure and exhibited no symptoms or unusual behavior or visual toxicity attributable to methanol poisoning. In an earlier study (Sayers et al, 1942), four dogs were exposed to airborne concentrations of methanol from 585 to 650 mg/m³, eight hours per day, seven days per week for 379 days in a continuously ventilated chamber. The authors performed a wide range of hematologic determinations and ophthalmoscopic examinations. No adverse effects of any kind were reported.

POTENTIAL REPRODUCTIVE, TERATOGENIC, OR CARCINOGENIC EFFECTS OF METHANOL EXPOSURE

Three studies have been reported in which the reproductive or teratogenic effects of methanol in nonprimate species were investigated.

Cameron et al (1984) exposed mature male rats (Sprague-Dawley) to airborne methanol concentrations of 260, 2,600, or 13,000 mg/m³ for one, two, four, or six weeks and examined them for alterations in circulating free testosterone, luteinizing hormone (LH), and follicle-stimulating hormone (FSH). Significantly decreased levels of circulating free testosterone were observed among rats exposed to 260 mg/m³ for two and six weeks and to 2,600 mg/m³ for six weeks. The high dose group (13,000 mg/m³) showed no change, however. The authors interpreted this as evidence that methanol exposure had lowered testicular production of testosterone. In addition, significant increases in circulating LH were observed after six

weeks of exposure to 13,000 mg/m³. No changes in FSH levels were observed.

Nelson et al (1985) administered 0, 6,500, 13,000, or 26,000 mg/m³ methanol to groups of approximately 15 pregnant Sprague-Dawley rats for seven hours per day on days 1 through 19 of gestation (for 26,000 mg/m³, days 7 through 15 only). The blood levels of methanol in the 26,000 mg/m³ group ranged from 8.34 to 9.26 mg/ml after one day of exposure and from 4.84 to 6.00 mg/ml after ten days of exposure.

The highest concentration of methanol produced slight maternal toxicity (unsteady gait) and a high incidence of congenital malformations, predominantly extra or rudimentary cervical ribs and urinary or cardiovascular defects. Among 15 litters exposed to 26,000 mg/m³, 14 contained at least one fetus with a skeletal malformation, and 10 contained at least one fetus with a visceral malformation. These incidences of malformation were significantly different from the control group ($p < .05$), which had no skeletal or visceral malformations in any of 15 litters. Similar malformations were seen in the group exposed to 13,000 mg/m³, but the incidence were not significantly different from those of controls. No increase in malformations was observed in the group exposed to 6,500 mg/m³, which the authors interpret as a no-effects level for this test system.

It was noted when reviewing this study that different incidences of visceral malformations were reported in the text than were reported in the accompanying tables. Those inconsistencies should be resolved before accepting the reliability of this paper. Moreover, the occurrence of maternal toxicity in the significantly affected group compromises an interpretation of the teratogenic effects as being solely the result of in utero methanol exposure.

Infurna and Weiss (1986) examined early behavioral development in Long-Evans rats exposed prenatally to methanol. The study focused on suckling and nest-seeking behaviors of the neonates. Treatment consisted of providing pregnant rats with a drinking solution containing 2% (volume ratio) methanol; one group received this treatment during gestational days 15 through 17, and a second group received the treatment during days 17 through 19. This treatment resulted in an average methanol consumption of 2.5 g/kg/day; controls received normal water. Increased latency to suckling behavior was observed in pups from both groups of methanol-treated dams, when the pups were tested 24 hours after their birth. In addition, pups in both groups exposed prenatally to methanol displayed a lower efficiency in seeking and reaching their home area when tested on postnatal day 10. Methanol treatments did not affect litter size, birthweight, weight gain during the preweaning period, infant mortality, or day of eye opening. Also unaffected were duration of gestation, weight gain in the third week of gestation, and maternal behavior on the day of parturition. The authors conclude that methanol "can be defined as a behavioral teratogen in rats, since no other signs of toxicity were apparent either in the mothers or the offspring."

The behavioral effects noted in this study occur at tissue levels of methanol lower than those associated with teratogenesis in the study by Nelson et al (1985), and may be of potential significance. However, maternal exposures to methanol during the three-day treatment periods in the Infurna and Weiss (1986) study (2.5 g/kg/day) are equivalent to at least 2,500 daily human exposures to methanol vapors under expected worst-case conditions. (As discussed in Section IV of this report, the added body burden of methanol resulting from worst-case exposure will be less than 1 mg/kg.) Clearly, dose-effect data on the parameters studied by Infurna and Weiss would help clarify whether or not humans may experience similar effects at or near expected ambient exposure levels. Such studies may be of particular value as they focus on endpoints representative of potentially subtle effects to the central nervous system.

CARCINOGENICITY AND MUTAGENICITY

There have been no studies reported in the peer-reviewed literature on the potential carcinogenicity of methanol in laboratory animals. As mentioned in Section III, the New Energy Development Organization (in Japan) sponsored chronic carcinogenesis bioassays in which mice (18 months) and rats (24 months) were exposed to 13, 130, and 1,300 mg/m³. The report issued from that study contains insufficient detail to allow for critical review.

Methanol has not been extensively tested for mutagenicity. It produced negative results in *Schizosaccharomyces pombe* with or without a microsomal activating system from mouse liver (Abbondandolo et al, 1980). Even with the addition of an activating system, this study did not properly address the mutagenicity of methanol metabolites. This is because the microsomal activating system used was from mice whose metabolic profile may be dissimilar to that in humans. In addition, soluble enzymes such as alcohol dehydrogenase, which are required in methanol metabolism in humans, are removed from microsomal preparations.

APPENDIX VI

EFFECTS OF FORMIC ACID (FORMATE)

Formic acid is the second oxidation product of methanol. It is a normal cellular constituent involved in numerous metabolic reactions. It is also a natural constituent of many foods. Formic acid is used in foods as a flavoring adjunct, brewing antiseptic, and preservative (FASEB, 1976). In a health evaluation conducted by FASEB (1976) for the Food and Drug Administration, the committee members concluded that there was no evidence that suggested that exposure to formic acid or sodium formate, when used as a food additive, would pose a hazard to the general public.

Effects Following Acute Formic Acid Exposure: Formic acid is a primary irritant that can cause severe damage to the skin, eye, or respiratory tract (Guest et al, 1982). Workers exposed to 15 ppm (28 mg/m³) of formic acid in the air have complained of nausea (ACGIH 1985b). Rats (Wistar) exposed to 38 mg/m³ of formic acid vapor 6 hours per day for 3 or 8 days were conspicuously inactive during the exposure period, but displayed no clinical signs of toxicity at the time of sacrifice (Zitting and Savolainen, 1980). Slight alterations in glutathione levels in the brains, livers, and kidneys of the rats were observed along with minor effects on drug metabolizing enzymes. At physiologic pH, formic acid dissociates to formate and a hydrogen ion. As discussed in the body of this report, high formate levels in the body have been associated with ocular toxicity in monkeys and humans after high methanol exposures.

Effects Following Repeated or Prolonged Formic Acid Exposure: Chronic administration of formic acid has not been shown to cause significant adverse effects in laboratory animals. Malorny (1969a) administered 0.2% calcium formate in the drinking water to rats (Wistar) for 3 years, or 0.4% calcium formate for 2 years, and no adverse effect on growth, fertility, or function in up to 5 generations was reported. Other shorter-term studies were cited by Guest et al (1982), in which the only effect reported was a decrease in the rate of body weight gain in rats. No chronic studies in non-human primates

are available. The occupational standard recommended by ACGIH (1985b) and adopted by OSHA for exposure to formic acid is 5 ppm (9 mg/m³), a level designated to protect workers from developing adverse health effects attributable to irritation of the eyes, respiratory tract, and skin.

Potential Reproductive, Teratogenic, Mutagenic, and Carcinogenic Effects of Formic Acid Exposure: Injection of 5, 10, or 20 mg of sodium formate into fertilized chicken eggs did not produce toxic or teratogenic effects (Malorny 1969a). In a 5-generation study that used Wistar rats given 150 to 200 mg of calcium formate per day (Malorny 1969a), no adverse effect on reproduction was noted.

Formic acid has been reported to be mutagenic in *Escherichia coli* and in *Drosophila* germ cells but no effect on DNA transformation in *Bacillus subtilis* was found (Guest et al, 1982). No carcinogenicity studies have been reported for formic acid. In the chronic study by Malorny (1969a), in which Wistar rats were administered calcium formate for 2 or 3 years at a level of 0.4% or 0.2% in the drinking water, no gross tumor formation was reported; tumor formation, however, may not have been an endpoint that was specifically evaluated.

In summary, there is substantial evidence to suggest that formic acid (formate) is the causative agent in methanol-induced metabolic acidosis and ocular toxicity. Formic acid is a primary irritant upon direct contact to the skin, eye, or respiratory mucosa. Systemically, formic acid has been shown to cause few adverse effects either after acute or chronic administration. It did not cause reproductive or teratogenic effects in fertilized chicken eggs or rats. The carcinogenic potential of formic acid has not been evaluated. It should be noted, however, that it was found to be mutagenic in some microbial test systems. The 1976 FASEB report concluded that exposure from the use of formic acid or sodium formate as a food additive posed no threat to human health. Thus, it appears that the formate-associated effects on the visual system following acute high-level exposure to methanol are the effects of greatest concern with respect to formic acid.

APPENDIX VII

Reprinted from: Tephly, T.R. and McMartin, K.E. Methanol Metabolism and Toxicity, in Aspartame: Physiology and Biochemistry, Stegink, L.D. and Filer, L.J., Jr., eds., Marcel Dekker, New York, 1984, pp. 111-140, by courtesy of Marcel Dekker, Inc.

Methanol Metabolism and Toxicity

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Metabolism of Methanol

Two enzymes are important in the oxidation of methanol to formaldehyde, alcohol dehydrogenase, and catalase (Fig. 2). The existence of relatively selective inhibitors for each enzyme has made it possible to test their importance in methanol oxidation in animals. It had been known for many years that the metabolism of methanol was blocked by the administration of ethanol and that methanol toxicity was attenuated by ethanol. Roe (8) suggested that humans who had taken ethanol simultaneously with methanol had less severe toxicity than when methanol was ingested alone. The assumption had existed for years that alcohol dehydrogenase was the major enzyme involved in methanol oxidation. Studies on alcohol dehydrogenase by Lutwak-Mann (52) showed that a partially

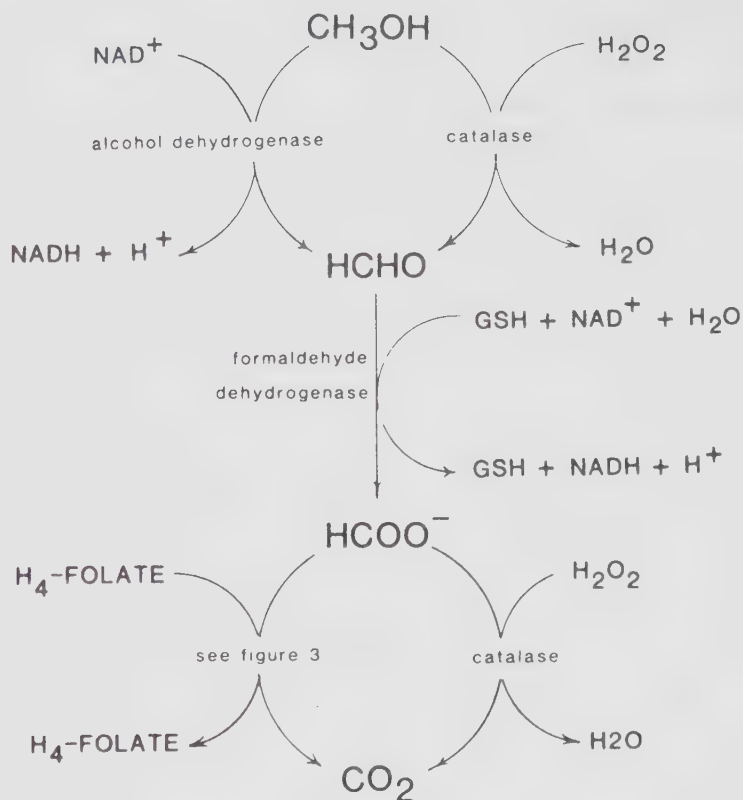


Figure 2 Biochemical reactions in the oxidation of methanol to carbon dioxide.

purified preparation of horse liver alcohol dehydrogenase oxidized methanol, although at a slower rate than ethanol. However, when crystalline horse liver alcohol dehydrogenase was prepared, it appeared to be incapable of catalyzing the oxidation of methanol (53,54), an observation that directed the attention of investigators to the catalase-peroxidative system as a mediator of the metabolism of methanol.

Interest returned to alcohol dehydrogenase and its role in methanol oxidation when Kini and Cooper (55) showed that, at high substrate concentrations, methanol was metabolized by crystalline horse liver alcohol dehydrogenase. Kini and Cooper (55) also showed that it was possible to copurify ethanol and methanol dehydrogenase activities from monkey liver. Their results conclusively demonstrated that alcohol dehydrogenase from monkey liver was capable of catalyzing methanol oxidation in vitro. Makar and Tephly (56) repeated these studies and showed that monkey liver alcohol dehydrogenase catalyzes methanol oxidation in vitro and that this activity is inhibited by the alcohol dehydrogenase

inhibitors pyrazole and 4-methylpyrazole. They reported that the Michaelis constant for methanol was about six times higher than that observed for ethanol, results similar to those found by Kini and Cooper, who had reported a K_m of 17 mM for methanol and 2.7 mM for ethanol. Makar and Tephly (56) observed K_m values of 20 mM for methanol and 3.2 mM for ethanol with the monkey liver enzyme. Pyrazole and 4-methylpyrazole were found to be competitive inhibitors when methanol and ethanol were utilized as substrates for monkey liver alcohol dehydrogenase. 4-Methylpyrazole yielded a K_i value of 9 μ M which was about one-fourth that observed for pyrazole. Makar and Tephly (56) also showed that 4-methylpyrazole had no inhibitory properties toward catalase activity of rat liver homogenates in vitro or in vivo. Pyrazole, on the other hand, inhibits hepatic catalase activity when injected in vivo (57). Other studies have shown that purified hepatic alcohol dehydrogenase from rats (58) and humans (59,60) catalyze methanol oxidation. Although the Michaelis constant of methanol for alcohol dehydrogenase appears to be relatively high (10-100 mM), concentrations of this magnitude (20-30 mM) can be achieved in vivo after drinking a sizable quantity of either methanol or ethanol.

The inhibition of methanol oxidation by ethanol does not necessarily mean that the alcohol dehydrogenase system functions for methanol oxidation in a given animal species. Catalase can mediate the oxidation of a variety of alcohols to their corresponding aldehydes in the presence of a hydrogen peroxide-generating source (61). A study performed by Keilin and Hartree (62), using purified catalase and various peroxide-generating systems, showed that methanol and ethanol were metabolized at similar rates. Both rates were more rapid than those obtained with alcohols possessing higher molecular weights. Thus methanol and ethanol had about equivalent reactivities with the catalase peroxidative systems, whereas propanol and butanol appeared to display lower substrate reactivity. In fact, Keilin and Hartree suggested (62) that the physiological function of catalase might be involved with the metabolism of certain alcohols. Previously, it had been presumed that the exclusive function of catalase in the living organism was to decompose hydrogen peroxide. An important understanding of how alcohols such as ethanol or methanol might react with catalase in the presence of hydrogen peroxide was provided by Chance (63). He showed that substrates for catalase peroxide (complex I) react with substrates such as methanol and ethanol and promote the decomposition of the catalase peroxide complex, the rate of which was dependent upon the rate of reactivity with the substrate and the catalase-hydrogen peroxide complex. Chance postulated that catalase could conceivably account for most of the metabolism of methanol in the animal organism in vivo (63).

Definitive studies on methanol oxidation in vivo began with the use of selective inhibitors. Heim et al. (64) discovered that the herbicide 3-amino-1,2,4-triazole could inhibit hepatic and renal catalase activity in rats when injected intraperitoneally. This provided a means to test the direct participation of hepatic

catalase in the oxidation of methanol in vivo. Aminotriazole has since been a very useful and effective substance for studying the role of hepatic or renal catalase in the oxidation of agents in vivo. It does not inhibit erythrocyte catalase activity, nor does it affect liver cytochrome c content, blood hemoglobin levels, or urobilinogen excretion (64).

Nelson et al. (65) showed that aminotriazole had no effect on ethanol elimination in the dog, although hepatic catalase activity was markedly reduced. Mannering and Parks (66) showed that aminotriazole inhibited hepatic catalase activity in rats in vivo and that, in livers from rats whose hepatic catalase activity had been reduced by 90%, a marked inhibition of methanol oxidation to formaldehyde was observed in vitro. When crystalline beef liver catalase was added to reaction mixtures employing homogenates of rat liver obtained from aminotriazole-treated animals, methanol-oxidizing capacity was restored to control values. These results indicated that hepatic catalase activity was important for methanol oxidation in vitro and, furthermore, that the rate-limiting step in the process was likely to be the capacity of the liver to generate hydrogen peroxide (66). Thus, when peroxide-generating systems were added to hepatic homogenates in addition to crystalline liver catalase, a marked stimulation of activity beyond control values was observed. Mannering and Parks (66) also employed aminotriazole in order to determine whether catalase participated in the metabolism of methanol by rats in vivo. However, they found that aminotriazole had no effect on the rate of disappearance of methanol from the blood of rats. This apparent discrepancy was later explained (67) on the basis that considerable amounts of methanol are eliminated via excretory routes, as well as by metabolism, at the high doses of methanol which were employed in their studies (3 g/kg). When [^{14}C]methanol oxidation was studied by measuring $^{14}\text{CO}_2$ formation in vivo in rats, aminotriazole treatment markedly inhibited the oxidation of methanol to CO_2 (67).

Further evidence for a catalase-peroxidative system functioning in the metabolism of methanol in rats was provided in studies where ethanol and 1-butanol were employed as alternate substrate inhibitors of methanol oxidation. Ethanol and methanol have about equal reactivities with catalase peroxide complex I, while ethanol is 6-10 times more reactive than methanol with alcohol dehydrogenase (68). Thus if catalase was functioning in the oxidation of methanol by the rat, one would have expected a 50% inhibition of methanol oxidation by ethanol, and, if alcohol dehydrogenase were functionary, a 90% inhibition would have been expected. Tephly et al. (67) showed that when equimolar doses of ethanol and methanol were injected into rats, a 50% decrease in the rate of methanol oxidation occurred. When 1-butanol, which has only a slight reactivity with the catalase-hydrogen peroxide complex I, was injected, only a very slight inhibitory effect on methanol oxidation in the rat occurred. These results are consistent with the concept that the catalase-peroxidative system is the major catalyst of methanol oxidation in rats. Similar conclusions have been reached in isolated perfused rat liver experiments (69).

Although the role of a catalase-peroxidative system for the metabolism of methanol in rats was clear, different results were obtained with monkeys. Makar et al. (70) showed that pretreatment of monkeys with 1 or 3 g/kg body weight of aminotriazole 1 hr prior to methanol injection did not inhibit the rate of methanol metabolism, although hepatic catalase activity in livers from monkeys was reduced to 10% of control values. Studies were also performed using substrate inhibitors. When equimolar doses of ethanol and methanol were injected in monkeys, an 80% inhibition of the rate of methanol oxidation was observed (70). When 1-butanol, which produced only a slight effect on methanol oxidation in the rat, was injected into monkeys along with [^{14}C]methanol, a 90% inhibition of methanol oxidation was observed. Butanol is a highly reactive substrate for alcohol dehydrogenase, and, if alcohol dehydrogenase were functioning, one would have expected a 90% inhibition of methanol oxidation by 1-butanol. These results support the concept that the catalase-peroxidative system is not functional in methanol oxidation in the primate and that the metabolism of methanol in the monkey is dependent on the activity of alcohol dehydrogenase.

Other evidence for a major role of alcohol dehydrogenase in methanol oxidation in the monkey was provided by Watkins et al. (42), who showed that pyrazole markedly inhibited methanol oxidation in the rhesus monkey. Although pyrazole rapidly inhibited methanol metabolism *in vivo*, there was a possibility that inhibition of hepatic catalase activity by a pyrazole metabolite could be responsible for the inhibition of methanol oxidation in the monkey. Thus 4-methylpyrazole, a more potent inhibitor of alcohol dehydrogenase activity than pyrazole and one which does not inhibit hepatic catalase activity (56), was tested in the monkey (34). 4-Methylpyrazole was found to be a potent inhibitor of methanol oxidation with little or no effect on hepatic catalase activity.

Thus a major role of alcohol dehydrogenase in the metabolism of methanol *in vivo* in the monkey has been established. McMartin et al. (34) also showed that 4-methylpyrazole prevents the development of methanol poisoning in the monkey.

The question of why the peroxidative system does not function in the monkey has been examined. It should be recalled that Mannering and Parks (66) showed that when a peroxide-generating system was added to rat hepatic homogenates, peroxide generation appeared to be a rate-limiting factor. When a glucose and glucose oxidase preparation was added, marked stimulation of methanol oxidation occurred. When catalase activity had been reduced markedly, such as from aminotriazole-treated rats, glucose and glucose oxidase addition did not stimulate methanol oxidation (66). Goodman and Tephly (71) suggested that the monkeys may not metabolize methanol through a catalase-dependent system owing to decreased activity levels of peroxide-generating enzymes. Since peroxide-generating systems appear to be rate limiting for methanol oxidation via a catalase-dependent system in the rat, these workers proposed that this system should be rate limiting in the monkey, perhaps to an even greater degree (71) than noted in the rat. It is well known that urate oxidase activity is essentially absent in human liver, and

Goodman and Tephly (71) have shown that urate oxidase activity was also very low in monkey liver. Furthermore, glycolate oxidase activity, xanthine oxidase activity, and other peroxide-generating enzyme activities are also very low in monkey and human liver (72). This could account for why methanol oxidation in the monkey via a catalase-peroxidative system is difficult to demonstrate. Makar and Mannering (58) also suggested that the catalase distribution in the cell may be a consideration.

A third possible mechanism by which methanol could be oxidized to formaldehyde has been suggested by Rietbrock et al. (73) and Teschke et al. (74). This system, the hepatic microsomal mixed-function oxidase system, employs the hepatic endoplasmic reticulum, NADPH, and molecular oxygen.

METABOLISM OF FORMALDEHYDE

Formic acid was considered as the toxic agent in the acidosis seen in methanol poisoning until Van Slyke and Palmer (11) discredited the toxic role of formate. They failed to account for the increased organic acid excretion observed in methanol toxicity as due to formate. Potts (31) also failed to account for the organic acids excreted in the urine as due to formate following methanol poisoning in monkeys. Thus formaldehyde became a candidate as a causative agent in the toxicity of methanol poisoning (41,75,76), even though no one had demonstrated the presence of elevated formaldehyde levels in body fluids or tissues following methanol administration. Keeser (77) appeared to demonstrate the presence of formaldehyde in the cerebrospinal fluid, vitreous humor, and peritoneal fluid of rabbits which had been administered methanol. However, these studies were rather incomplete, lacked appropriate controls, and the method employed to measure formaldehyde lacked sensitivity and specificity. No formaldehyde could be detected in blood, urine, or tissues obtained from methanol-intoxicated animals in studies performed by Koivusalo (51) and Scott et al. (30) or from methanol-poisoned humans (8,78).

There are several ways by which formaldehyde can be disposed of in biological systems. First, formaldehyde has a high degree of reactivity with proteins and other endogenous compounds containing active hydrogen atoms (79). Formaldehyde can combine with any number of functional groups found in proteins or nucleic acids. Thus it may immediately form adducts with cellular constituents, leading to the formation of stable intermediates.

Strittmatter and Ball (80) isolated a formaldehyde-specific, NAD-dependent formaldehyde dehydrogenase from beef liver in 1955 and pointed out that this enzyme required reduced glutathione (GSH). This enzyme, which appears to be quite specific for formaldehyde, is often isolated with glutathione thiolase (81, 82). In the reactions catalyzed by this enzyme (Fig. 2), formaldehyde combines with reduced GSH to form S-formyl glutathione, and in the presence of the thiolase, the product hydrolyzes to form formic acid and reduced glutathione. Reduced glutathione is therefore a key agent in the generation of formate from

formaldehyde. The first reaction appears to be freely reversible, but the second reaction is not, a feature which explains the apparent irreversibility of the two-step reaction as described by Strittmatter and Ball (80). Formaldehyde dehydrogenase activity is present in rat liver, human brain, and a number of other species and tissues such as retina (83). These tissues have not been examined adequately for the presence of S-formyl glutathione hydrolase. The specific activity of this enzyme in crude preparations appears to be quite high, and its presence would be expected in other tissues (81).

Formaldehyde oxidation can also occur in liver mitochondria through an aldehyde dehydrogenase activity (or activities) which is likely to be similar to the aldehyde dehydrogenases of mitochondria that have been described previously (84-86). Aldehyde dehydrogenase activity of mitochondria appears to be very high and is capable of reacting nonspecifically with many aldehyde substrates. Thus it is likely that formaldehyde-oxidizing capabilities of liver are extremely high, either through the formaldehyde dehydrogenase-S-formyl glutathione hydrolase system or through aldehyde dehydrogenase activities in mitochondria or cytosol. Goodman and Tephly (87) have shown that the formaldehyde dehydrogenase activity of human liver is, in fact, higher than that of rat liver. Thus one cannot explain, at this time, the fact that methanol poisoning is uniquely present in humans or monkeys on the basis of an inability to metabolize formaldehyde, since the conversion of formaldehyde to formate can apparently proceed as readily in humans as it does in rats.

Formaldehyde can be metabolized through the tetrahydrofolic acid-dependent one-carbon pool which is capable of utilizing one-carbon units at various oxidation levels and transferring these one-carbon moieties to various endogenous acceptors. Apparently, free formaldehyde enters these reactions by combining with tetrahydrofolate nonenzymatically (88) or through the formaldehyde-activating enzyme to form N⁵,N¹⁰-methylenetetrahydrofolate. This enzyme has been demonstrated in pigeon liver by Osborn et al. (89) and has been found to be present in a number of mammalian tissues (90).

The metabolism of formaldehyde has been studied by Malorny et al. (91) in dogs and cats *in vivo*. These investigators administered formaldehyde intravenously and orally to dogs and showed that there was a rapid appearance of formic acid in blood plasma and the presence of only negligible levels of formaldehyde in blood. Experiments *in vitro* with human blood showed that formaldehyde was oxidized to formic acid (92,93). Rietbrock (94) showed that in dogs, cats, rabbits, guinea pigs, and rats the infusion of formaldehyde resulted in a rapid disappearance of formaldehyde from the blood with a half-life of approximately 1 min. Malorny et al. (91) found that when equimolar amounts of formaldehyde, formic acid, or sodium formate were infused in dogs, the peak concentrations of formic acid in the plasma were equivalent in all three cases, indicating that formaldehyde was rapidly metabolized to formic acid.

Although it is possible that formaldehyde may be responsible for certain of

the toxic findings in methanol poisoning, it would be unlikely that it could account for the metabolic acidosis, since formate appears to be the major factor in the metabolic acidosis seen in monkeys and humans poisoned with methanol. It is also unlikely that formaldehyde can be generated in the liver and delivered to the optic nerve in an intact state. Therefore either formaldehyde forms a product with some endogenous acceptor which is responsible for the ocular toxicity, or formaldehyde is generated in situ in the eye, where it may exert an effect on the ocular system. Although these possibilities cannot be ruled out at this time, the responsibility of formaldehyde for the ocular toxicity of methanol is unlikely, since formate itself can produce ocular toxicity in the monkey (95). In studies where blindness in monkeys was produced from formate, no formaldehyde could be detected in body fluids or tissues (95). In any case, more studies need to be performed on the fate of formaldehyde in the organism in order to disregard it as a toxic agent in the methanol poisoning syndrome in man.

FORMATE METABOLISM

Nonprimates

The ability of animal tissues to oxidize formate into CO_2 was first reported by Batelli (96) and Battelli and Stern (97), who observed that tissues obtained from a variety of animals, such as the horse, cow, sheep, dog, and rabbit, were capable of oxidizing formate into CO_2 in the presence of hydrogen peroxide (Fig. 2). More than 40 years later, Chance (63) studied the kinetics of the catalase-hydrogen peroxide system with different substrates and showed that formate reacts with the hydrogen peroxide catalase complex (complex I).

In subsequent years, a number of *in vitro* investigations strongly indicated a key role of the catalase-hydrogen peroxide system in the oxidation of formate. Some of the experimental results leading to this conclusion are the following:

1. There is a good correlation between the formate-oxidizing ability and the catalase activity in liver preparations of different species (98), in different tissues within one animal species (99), and in the subcellular compartments from tissue preparations (100).
2. Administration of aminotriazole to guinea pigs greatly lowered the formate-oxidizing ability of liver fractions *in vitro* (98).
3. Certain types of neoplasms in rats (101), mice (102), and humans (103) lead to a marked lowering of both catalase activity and the formate-oxidizing ability *in vitro*.
4. Folate-deficient rats possess a marked impairment in formate-oxidizing ability (104) and lowered hepatic catalase levels.
5. Decreased formate oxidation *in vitro* results from decreased hydrogen peroxide generation caused by factors such as a decreased hepatic xanthine oxidase activity, vitamin B_6 deficiency (105), or thyrotoxicosis (100). On the

other hand, factors that increase hydrogen peroxide generation stimulate formate oxidation. This can be accomplished by supplementing liver preparations with hypoxanthine, a known substrate of xanthine oxidase.

Another path of formate oxidation to CO_2 is the folate biochemical pathway (88,90,106,107). Formate enters into the folate pool by combining with tetrahydrofolate (THF) to form 10-formyl-THF, a reaction catalyzed by 10-formyl-THF synthetase, an enzyme widely distributed among mammalian tissues (108). Kutzbach and Stokstad (109) showed that 10-formyl-THF oxidoreductase catalyzes the oxidation of the formyl group directly to CO_2 . Thus there is a two-step conversion of formate to CO_2 .

Rietbrock et al. (73) suggested that exogenously administered formate, or formate arising from methanol metabolism *in vivo*, is oxidized via the folate-dependent pathway. They found an inverse correlation between plasma levels of folate in different animal species and the half-life of exogenously administered formate. They also reported that dogs accumulated formic acid to a small extent (2 mEq/liter) in their blood following methanol administration. Pretreatment of dogs with folic acid prior to methanol produced a lower blood formate level, whereas methotrexate (an inhibitor of dihydrofolate reductase) had the opposite effect (110).

Palese and Tephly (111) measured $^{14}\text{CO}_2$ formation following [^{14}C] formate administration to rats and showed that folate deficiency resulted in a greatly diminished rate of formate oxidation. In contrast, administration of aminotriazole, the potent catalase inhibitor, did not inhibit the rate of formate oxidation to CO_2 . Administration of ethanol in molar ratio of 22:1 (ethanol:formate) did not alter the rate of formate oxidation in the rat. However, in folate-deficient rats, the catalase-hydrogen peroxide system may serve as an alternate pathway, since, in folate-deficient rats aminotriazole or ethanol administration did result in some inhibition of the rate of formate oxidation (111).

The knowledge that formate is being metabolized *in vivo* via a folate-dependent system has been utilized to advantage in order to produce metabolic acidosis in rats after methanol treatment. Rats, made folate deficient, oxidize formate at a markedly slowed rate (111,112), and administration of methanol (4 g/kg) to folate-deficient rats leads to high formate levels and severe metabolic acidosis (113). Blood formate levels reached values as high as 18 mEq/liter in these animals. This value is higher than blood formate levels noted in methanol-poisoned monkeys (34).

Monkeys

In monkeys the folate-dependent pathway is also the major route of formate oxidation to CO_2 . Makar et al. (70) showed that aminotriazole had no effect on methanol oxidation to CO_2 in the monkey, and McMartin et al. (114) demonstrated that neither the rate of formate oxidation nor the half-life of formate in

the blood was altered by aminotriazole. However, the rate of formate metabolism in folate-deficient monkeys was approximately 50% lower than that observed in control monkeys. Formate oxidation was stimulated in monkeys by the administration of either folic acid (114) or 5-formyl-THF (115).

McMartin et al. (114) also showed that the sensitivity of monkeys to methanol was related to folate, since folate-deficient monkeys became especially sensitive to the toxicity of methanol relative to the amounts of formate produced. Thus, when 0.5 g/kg of methanol was given to either folate-deficient or control monkeys, the level of blood formate in the folate-deficient animals was more than two times greater than that observed in the control animals.

Noker and Tephly (115) then showed that methanol toxicity can be modified considerably in monkeys by the administration of folate derivatives. These workers followed the course of methanol toxicity in monkeys administered [^{14}C]methanol (2 g/kg) or [^{14}C]methanol with repetitive doses of 5-formyl-THF. In monkeys treated with 5-formyl-THF (2 mg/kg at 0, 4, 8, 12, and 18 hr after methanol), blood formate levels were significantly decreased (by at least 50%) from those observed in the untreated animals. Similar results were obtained when sodium folate was employed instead of 5-formyl-THF. In both treated and untreated monkeys, the elimination of methanol from blood followed zero-order kinetics and proceeded at a rate of 7.9 mg/dl per hour in the 5-formyl-THF-treated animals, and at 7.1 mg/dl per hour in the untreated animals. Therefore the clearance of methanol was not altered by folate administration. In addition, the distribution and route of metabolism of [^{14}C]methanol did not appear to be changed by 5-formyl-THF treatment, since the total amount of ^{14}C label recovered in urine as either expired [^{14}C]methanol or $^{14}\text{CO}_2$ was the same for both treated and untreated monkeys. However, the rate of methanol oxidation to CO_2 was significantly increased in those animals treated with 5-formyl-THF, and folate treatment was effective in reducing blood formate levels by increasing the rate of formate metabolism to CO_2 . Blood pH and blood bicarbonate levels remained within the normal range in animals treated with 5-formyl-THF, in contrast to the marked bicarbonate depletion, high blood formate levels, and metabolic acidosis observed in animals not given 5-formyl-THF.

Noker and Tephly (115,116) have also shown that 5-formyl-THF (when given in repetitive doses) is effective in reversing methanol toxicity in the monkey once it has developed. The accumulation of blood formate in monkeys could be markedly altered by 5-formyl-THF, even when administered after toxicity became apparent. A rapid decline in blood formate levels was observed in methanol-poisoned animals several hours after the initiation of 5-formyl-THF treatment. In monkeys not given 5-formyl-THF, formate levels continued to climb. The decline in formate concentrations in monkeys treated with folate was coupled to an increase in the rate of CO_2 formation from methanol.

The results demonstrate that the severity of methanol toxicity in monkeys is correlated with accumulation of formate in the blood and that this can be sig-

nificantly modified by procedures which provide the monkey with more folate. These results suggest that there is a reciprocal relationship between the formate oxidation rate and the hepatic folate level of the animal. They suggest the possible use of folates for the treatment of human methanol toxicity.

Regulation of Formate Oxidation Through Regulation of Tetrahydrofolate

Since the folate biochemical pathway is primarily involved in the metabolism of formate, the regulation of the rate of formate metabolism is governed by the regulation of the hepatic tetrahydrofolate concentrations in liver. This concept has been advanced recently by studies which have explored the role of 5-methyl-THF:homocysteine transmethylase (methionine synthetase). This cytosolic enzyme is responsible for the methylation of homocysteine to form methionine as well as for the conversion of 5-methyl-THF to THF (Fig. 3). It requires methyl-

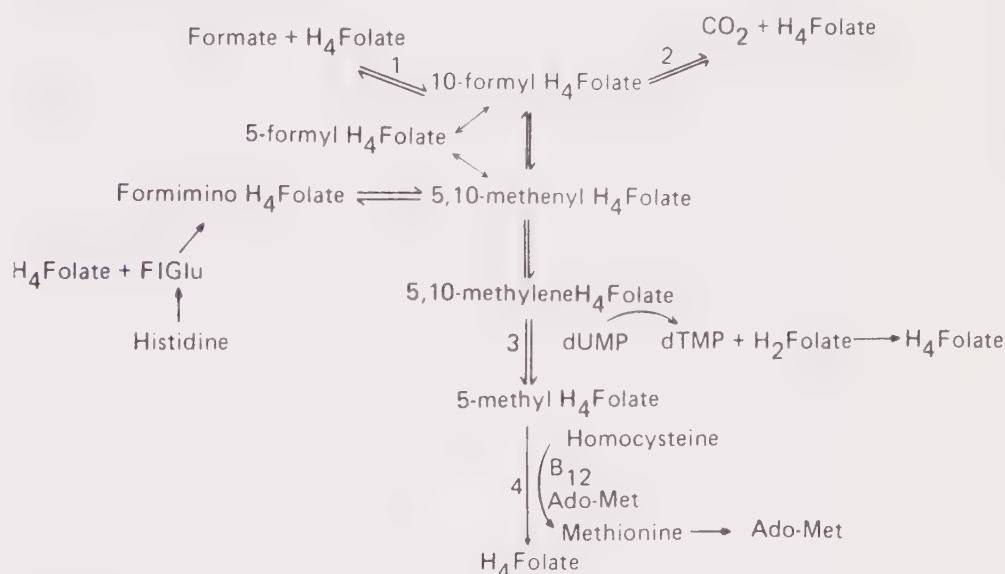


Figure 3 Pathway of folate-dependent formate metabolism (H₂ folate, dihydrofolate; H₄ folate, tetrahydrofolate; B₁₂, vitamin B₁₂; Ado-Met, S-adenosylmethionine). Reaction 1 is catalyzed by formyl-tetrahydrofolate synthetase and requires activation of formate by ATP. Reaction 2 is catalyzed by formyl-tetrahydrofolate dehydrogenase and utilizes NADP⁺. Reaction 3 is catalyzed by methylene-tetrahydrofolate reductase and is thought to be irreversible. Reaction 4 is catalyzed by methyl-tetrahydrofolate homocysteine methyltransferase (methionine synthetase) and is dependent upon vitamin B₁₂ and catalytic amounts of adenosylmethionine, a reducing system.

cobalamin and S-adenosylmethionine for maximal activity. As far as we know, methionine synthetase is the only methylcobalamin-dependent enzyme in the mammalian organism. The anesthetic gas nitrous oxide has been reported to react with transition methyl complexes, such as the cobalt-ligand complex in vitamin B₁₂, and oxidizes the coenzyme from the active cob(I)alamin form to the inactive cob(III)alamin form (117). Deacon et al. (118) have shown the inhibition of hepatic and brain methionine synthetase activity *in vivo* by nitrous oxide, and Eells et al. (119,120) demonstrated that, following nitrous oxide treatment of rats, there was a significant decrease in hepatic levels of tetrahydrofolate forms and an increase in hepatic 5-methyl-THF. Rats treated with nitrous oxide also exhibited a marked decrease in the rate of formate oxidation to carbon dioxide. When methanol (4 g/kg) was administered to rats which were exposed to nitrous oxide:oxygen (50:50) for 2 hr, there was a marked metabolic acidosis in these animals, with accumulation of blood formate, a decrease in blood pH to 7.2, and a depletion of blood bicarbonate. This metabolic acidosis produced after the administration of methanol to rats had not been demonstrated previously, except where rats were made folate deficient (104). Hepatic methionine synthetase activity was reduced to 10% of control levels in animals treated with N₂O:O₂ (50:50), a finding which accounts for the depletion of hepatic tetrahydrofolate. Recently, Eells et al. (120) demonstrated an excellent correlation between the rate of formate oxidation in rats with hepatic tetrahydrofolate levels. Since S-adenosylmethionine levels are also dependent upon hepatic methionine levels, one would expect alteration of S-adenosylmethionine concentrations in liver. S-Adenosylmethionine levels are depleted by the treatment of rats with nitrous oxide, and a good correlation between tetrahydrofolate levels and S-adenosylmethionine was also recorded (120).

Methionine administration to rats which have been treated with nitrous oxide leads to a reversal of the depletion of tetrahydrofolate levels in liver and a reversal of the inhibition of formate oxidation produced by nitrous oxide (120). However, the mechanism by which methionine is capable of reversing the depletion of tetrahydrofolate brought on by nitrous oxide treatment is still unexplained; that is, although nitrous oxide inhibits methionine synthetase activity and depletes tetrahydrofolate levels, methionine administration does not reverse the inhibition of methionine synthetase activity, although it restores tetrahydrofolate in liver. Therefore methionine cannot be exerting its effect by a direct action on methionine synthetase activity. It is possible that methionine exerts its effect through the elevation of S-adenosylmethionine concentrations in liver. Following methionine treatment, there is a marked elevation of S-adenosylmethionine levels in rat liver (120) and S-adenosylmethionine acts as an inhibitor of 5,10-methylene-THF reductase (121). More work is needed in order to determine the mechanism by which methionine exerts its reversal of the nitrous oxide depletion of hepatic tetrahydrofolate.

Recent studies in our laboratory have shown that treatment of monkeys with a nitrous oxide:oxygen (50:50) mixture leads to marked sensitization of the monkey to methanol toxicity. Following a dose of 1 g/kg of methanol (a dose which produces only a slight increase in blood formate in monkeys), there was a marked accumulation of formate (4 mEq/liter) 12 hr after methanol. These values are greater than blood formate levels observed when 2 g/kg of methanol were given to air-breathing monkeys.

A great deal more work is needed in order to understand which step of the many enzymatic reactions in the folate biochemical pathway regulates the regeneration of tetrahydrofolate in monkeys. However, it is important to realize that primates are at some risk with respect to their folate regulation; and it would appear to be important for future work to determine that step or process which is deficient and which places the primate at a distinct liability when it comes to the disposition of one-carbon moieties.

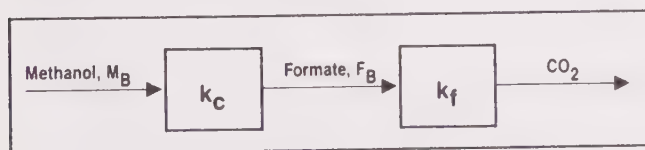
APPENDIX VIII

KINETIC MODEL OF FORMATE ACCUMULATION FOLLOWING ACUTE METHANOL EXPOSURE

The following two-compartment model may be used to estimate blood formate levels following brief (less than 15 minutes), low-level exposures to methanol. Since blood clearance of methanol proceeds with about a 3-hour half-time, such brief exposures can be considered as a single acute dose. The model assumes:

1. All metabolic pathways remain in a first-order domain.
2. Formate distributes relatively evenly to body water, and renal excretion of formate is negligible compared to metabolism.
3. The entire dose of methanol enters the metabolic pathway.

Consider the following system:



Let

t = time post-ingestion (hours)

M_B = aqueous concentration of blood methanol (mM, i.e., millimoles per liter) and, $M_B = M_{B0}$ at $t = 0$.

F_B = aqueous concentration of blood formate (mM)

k_c = clearance rate constant of methanol into the metabolic pathway

k_f = rate constant of formate metabolism

Then, using first-order principles,

$$(1) \frac{dF_B}{dt} = \frac{-dM_B}{dt} - k_f F_B \text{ and since, } (2) M_B = M_{B0} e^{-k_c t},$$

$$(3) \frac{dM_B}{dt} = -k_c M_{B0} e^{-k_c t}$$

Therefore,

$$(4) \frac{dF_B}{dt} + k_f F_B = k_c M_{B0} e^{-k_c t}$$

Multiplying both sides by $e^{k_f t}$ yields

$$(5) e^{k_f t} \frac{dF_B}{dt} + k_f F_B e^{k_f t} = k_c M_{B0} e^{(k_f - k_c)t}$$

OR

$$(6) \frac{d(F_B e^{k_f t})}{dt} = k_c M_{B0} e^{(k_f - k_c)t}$$

Integrating and setting $F_B = 0$ at $t = 0$ yields

$$(7) F_B = \frac{k_c}{k_f - k_c} M_{B0} (e^{-k_f t} - e^{-k_c t})$$

and, setting dF_B/dt equal to zero solves for the time, t_{\max} , at which F_B is maximized,

$$(8) t_{\max} = \frac{\ln(k_c/k_f)}{k_c - k_f}$$

Finally, the ratio of the maximized concentration of formate to the initial concentration of methanol is

$$(9) \frac{F_{B\max}}{M_{B0}} = \frac{k_c}{k_f - k_c} (e^{-q} - e^{-r})$$

$$\text{where, } q = \frac{k_c}{k_c - k_f} \ln(k_c/k_f), \text{ and } r = \frac{k_f}{k_c - k_f} \ln(k_c/k_f)$$

This value depends only on the ratio of k_f to k_c (as does the "dimensionless time" $k_c t_{\max}$).

Thus, one may easily project blood formate levels using measured values of k_c and k_f , as in the following example:

A worst-case exposure in a hot-soak garage produces a methanol body burden of 1 mg/kg, which is equivalent to 0.05 mM (M_{B0}). Methanol clears from the bloodstream into the metabolic pathway with a $T_{1/2}$ of 3 hours, meaning $k_c = 0.693/3 = 0.23 \text{ hr}^{-1}$, and formate clearance proceeds with a $T_{1/2}$ of 45 minutes or $k_f = 0.92 \text{ hr}^{-1}$.

Applying formulas (7) and (8) yields $t_{\max} = 2.0$ hours, at which time $F_{B\max} = 0.0082 \text{ mM}$. Since measured levels of background formate are about 0.2 mM, the maximal increment of formate amounts to 4% of background. For the example described, Figure VII-1 displays the time courses that equations (2) and (7) predict for methanol and formate, respectively. Using the example's clearance characteristics as a point of reference, Table VII-1 (center box is reference) shows the relative values of t_{\max} and F_B as $T_{1/2}$ for methanol and formate vary. The table shows the expected: as the efficiency of formate metabolism decreases relative to methanol clearance, formate accumulates to a greater degree. In addition, formate peaks, for all cases shown, within 1.3 to 2.7 hours of exposure. However, all the curve shapes remain similar to the one plotted for the example.

		T _{1/2} Methanol (hour)		
		2	3	4
T _{1/2} Formate (min)	30	1.00 0.67	0.74 0.78	0.59 0.86
	45	1.32 0.85	Fmax 1.00 1.00 tmax	0.81 1.11
	60	1.59 1.00	1.22 1.19	1.00 1.33

upper left = maximal formate concentration relative to base case
lower right = time to achieve maximal formate relative to base case

Table VIII-1 Relative values of (1) peak blood formate level and (2) the time from methanol exposure to achieve that level as a function of the blood clearance half-times for methanol and formate. In reference condition (middle cell), half-time for methanol clearance is 3 hours and for formate is 45 minutes. Upper left of each cell is relative blood formate level; lower right is relative time to achieve maximal concentration.

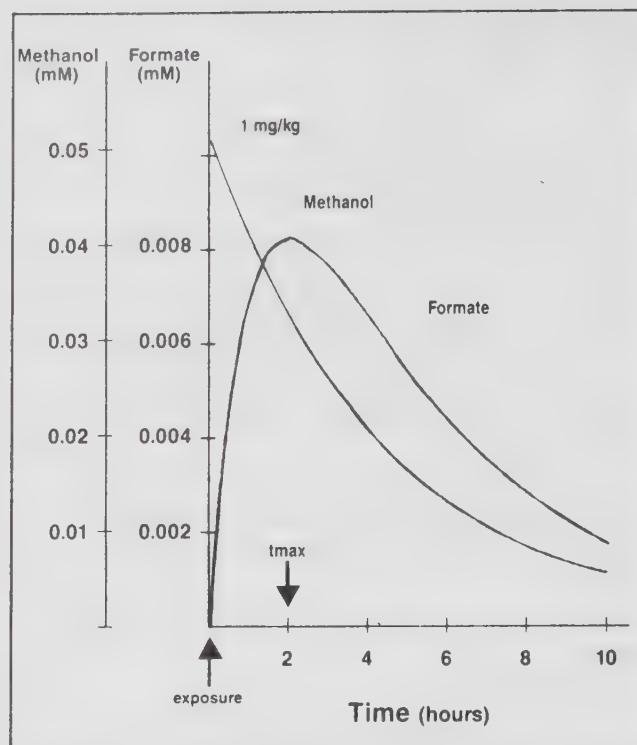


Figure VIII-1: Blood levels of methanol and formate following an initial body burden of 1 mg/kg methanol according to the two-compartment model presented in the text of Appendix VIII. Clearance half-times are 3 hours for methanol and 45 minutes for formate.

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A NOTE ON PROCESS

In the spring of 1984, HEI held an initial seminar in Albuquerque, New Mexico to examine the issue of methanol vapor emissions from motor vehicles. The workshop led HEI to commission an overview paper by Dr. Thomas Tephly, an internationally known expert in this area. This paper, which was completed in late 1984, was an essential aid to the Institute's Health Research Committee in defining areas of further inquiry. The staff of the Institute, operating at the direction of the Health Research Committee, then contracted with the Environ Corporation, a health and environmental consulting firm in Washington, D.C., to pull together the published literature on the subject. This report, which was completed in the winter of 1986, formed the basis for another review of the subject at the Health Research Committee's spring 1986 meeting. At that meeting, the Health Research Committee requested the HEI Board of Directors to ask the Administrator of the Environmental Protection Agency (EPA) for his views on the priority of this issue from his perspective. A May, 1986 letter from HEI's chairman did this. Further, at HEI's request, representatives from EPA's Office of Health Research and Office of

Mobile Sources attended the Health Research Committee's June 1986 meeting to discuss this issue. Both in person and in a letter from EPA's assistant administrator for research and development, the EPA reaffirmed the high priority of its interest in the development of methanol fuel. Accordingly, the Health Research Committee recommended to the Board that it authorize an analysis that would present HEI's sponsors and the public with a careful appraisal of the current health evidence and its implications, as well as research opportunities that could be implemented by the scientific community, including HEI.

The Health Research Committee wishes to acknowledge and thank a number of contributors to the development of this report, including the Environ Corporation, Dr. Morton Grant, Dr. Dag Jacobsen, Dr. David Leith, Dr. Kenneth McMartin, Dr. Thomas Tephly, Dr. Peter Valberg, and Dr. Myron Wolbarscht. Ms. Jessica Schwartz edited this document. The Committee would like to join the Board in congratulating Dr. Robert Kavet for his outstanding work as primary author of this analysis.

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Formation and Control of Aldehydes in Alcohol Fueled Engines

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Formation and Control of Aldehydes in Alcohol Fueled Engines

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ABSTRACT

Aldehyde formation and emissions from alcohol fueled engines are studied and presented in this paper. Several chemical kinetic models on the mechanism leading to aldehyde formation have been examined to explore the appropriate control methods to reduce exhaust aldehyde emissions. Control of aldehydes in exhaust emissions by suitable alteration of engine operating parameters, by in cylinder treatment with additives like aniline and water, by external treatment like airpreheating, secondary air injection cooling water rate and exhaust treatment are examined. The concept of surface ignition for alcohol fuels is briefly presented as a long range objective for using alcohols with minimal aldehyde emissions.

THE RECENT ENERGY CRISIS has stimulated interest in alternative fuels for transportation sector. Among the various alternative fuels proposed, alcohols - especially methanol and ethanol - have received considerable attention as motor fuels or as gasoline blending components. It should however be noted that use of alcohol as engine fuel is as old as the engine itself, but the recent research efforts emphasize the emission aspects besides the performance and energy economy.

Both methanol and ethanol can be produced from renewable and nonrenewable resources. Various studies have shown the potential advantages of alcohol to power internal combustion engines from the point of view of power, energy economy, cleanliness and freedom from excessive harmful emissions to atmosphere. These studies also point out that certain

disadvantages as compared to gasoline could also be overcome in the foreseeable future.

In recent years, much attention has been given to the understanding and minimizing of exhaust emissions. Most of this effort has been concerned with nitric oxide, carbon monoxide and unburned hydrocarbon emissions. Exhaust emission tests with methanol and ethanol (1,2,3,4)* compared to gasoline at the same compression ratio (CR) reveal considerable reduction in nitrogen oxide emission (NO_x) while

carbon monoxide (CO) and unburned hydrocarbon emissions (UBF) are at nearly the same level. On the other hand, aldehyde emissions increase enormously from two to ten times. Aldehyde emissions are only a small and relatively insignificant part of gasoline exhaust and therefore currently unregulated from the point of view of atmospheric air pollution. However, considering the large magnitude of aldehyde emission with alcohol exhaust, these emissions have to be controlled in such engines.

Studies of oxidation of different fuels at relatively low temperature have shown that reaction proceeds through various intermediate stages. With hydrocarbon fuels, the mechanism in the intermediate stages is through two parallel paths (4), of which only one involves the formation of aldehydes. On the otherhand, with alcohol fuels, the intermediate route is always through the formation of aldehydes formaldehyde in the case of methanol and principally acetaldehyde in the case of ethanol oxidation (4). This explains why aldehyde emissions are greater with alcohol fuels. It is important, therefore, to understand the formation processes of

*Numbers in the paranthesis indicate the references at the end of the paper.

aldehydes and what methods can be used to reduce them before alcohol fueled vehicles become of widespread use.

FORMATION OF ALDEHYDES IN INTERNAL COMBUSTION ENGINES

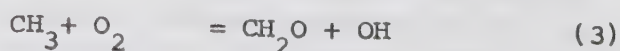
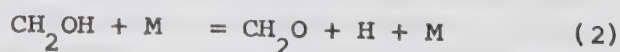
Measurement of aldehyde in the end gas region in spark ignition engines has been reported by Ricardo (6) in the early 1930's. Formaldehyde was always found in the non-flamed gases (7) under knocking conditions. These gases are not responsible for exhaust aldehydes as they are consumed by the advancing flame front.

Recent computer studies (8, 9) indicate that aldehydes do not survive in the bulk gas. It is now established that exhaust aldehydes are in addition formed as intermediate species of the oxidation of unburned methanol in the exhaust. There are many reasons why unburned fuel is found in the engine exhaust. It is found in the quench zone of the combustion chamber as the flame is extinguished against the cold cylinder walls. Fuels may also hide in the crevices of the chamber wall escaping combustion. It is to be recognised (10) that bulk of the liquid alcohol is inducted into the combustion chamber before evaporation and subsequently spread over the chamber walls to evaporate during the exhaust stroke. Browning and Pefley (11) used a detailed kinetic reaction mechanism to study aldehyde formation in the unburned quench layer under variety of conditions. Aldehydes were found to increase an order of magnitude over bulk gas concentrations as the flame quenched but this concentration was insufficient to account for measured aldehydes. A flow reactor study showed that additional aldehydes being formed from the unburned quench layer as it mixed with hot exhaust gases during the expansion and exhaust strokes. In their subsequent studies they (12) showed that aldehyde formation was not completely accounted by cylinder wall quenching as post flame diffusion removed most of the unburned fuel from quench layer at a fast rate without formation of significant aldehydes. The same authors followed up this work recently in a more exhaustive modelling (13) of the exhaust port and manifold with detailed chemical kinetics and one dimensional fluid mechanics in order to study the aldehyde formation during the exhaust stroke. Alde-

hyde formation and emissions were examined analytically for a variety of engine operating conditions including variation of equivalence ratio, compression ratio, spark timing and speed. A companion paper (14) described another chemical kinetic model to explain the mechanism leading to formaldehyde in a methanol fueled spark ignition engine. The model scheme included the reaction $\text{NO} + \text{HO}_2 = \text{NO}_2 + \text{OH}$ which became important in an exhaust system containing a large amount of nitric oxide under a relatively low temperature ranging from 600-1000° K. The model was also validated by experiments using a heated tube reactor fitted to the exhaust port. Computed results of the concentration of unburned methanol and formaldehyde compared well with the measured values in the reactor tube under various conditions of temperature and residence time.

Methanol was found to oxidise with increase in temperature and the concentration decreased with increase in residence time. Formaldehyde levels increased as oxidation of unburned methanol progressed and after reaching a maximum decreased with increase in residence time.

While the detailed chemical kinetic reaction mechanism for the oxidation of methanol is available in literature, the reaction scheme for ethanol to the best knowledge of the authors is not available. The classic mechanism of the methanol oxidation proposed by Bowman and Westbrook et al have been quoted and used by the authors of reference 13 and 14. From these references, there are two primary paths for the formation of formaldehyde, one through hydroxymethyl radicals CH_2OH and the other through methyl radical CH_3 . The following reactions account for formaldehyde formation and destruction at temperatures encountered in the combustion chamber and exhaust systems. Formation Reactions:



of these reactions, reaction (1) seems to account for almost all the consumption rate of CH_2OH in the temperature range prevailing² in the exhaust.

Dominant destruction reactions are



These results indicate that the presence of molecular oxygen in the exhaust gases might have a favourable effect on formaldehyde formation, while the absence of hydroxyl radical and atomic hydrogen might have unfavourable effect on formaldehyde destruction.

The foregoing basic work helps in the understanding of conflicting experimental results presented here and is also a necessary precursor to finding a solution to control methods.

EXPERIMENTAL

The following objectives were specified for experimental work. (a) Influence of engine operating parameters on aldehyde emission level - Effects of speed, load, spark-timing. equivalence ratio and compression ratio. (b) Influence of fuel treatment - An approach to destroy the aldehydes by the addition of aniline or water blending. (c) Control methods for aldehyde emissions - Additives, air pre-heating, secondary air injection cooling water regulation, after treatment of exhaust and new design aspects.

Test were conducted on single cylinder engines as these engines permitted flexibility for independently controlling the operating parameters without mixture maldistributions. Two engines were selected with the specifications given in Table 1 and 2.

Table - 1 - ENGINE 'A'

Make	- Dipco - Kirloskar (India)
Type	- Basic single cylinder diesel engine with a squish bowl in the piston, modified as spark ignition engine with the carburettor. A modified combustion chamber in the cylinder head permitted flexibility to operate the engine with

- the surface ignition facility. (Details are reported in Ref.27 and 28).

Compression Ratio	-16 (original) Variation from 7.8 to 12 made possible by modifications.
Bore	80 mm
Stroke	110 mm
Power rating	6.8 K.W.
Test Speed	1500 RPM
Displacement	0.553 Litre
Dynamometer	Electrical Torque reaction type.

Table 2 - ENGINE 'B'

Make	Waukesha Motor Co., USA
Type	Variable Compression - Ratio, ASTM-CFR engine with removable dome head (RDH)
Bore	96.8 mm
Stroke	92.1 mm
Displacement	0.678 Litre
Power rating	2.8 - 3.1 K.W.
Test Speed	1500 RPM
Spark timing	MBT
Dynamometer	Electrical torque reaction type

The following fuels were used without preheat for the studies. 100 per cent Methanol; 90 per cent Methanol + 10 per cent Water (by volume); 99 per cent Methanol + 1 per cent Aniline (by volume).

TEST CONDITIONS - In tests on both the engines, the operating conditions were maintained as required by the respective test objectives. For determination of aldehyde the standard MBTH method (15) was used. Two sampling techniques were adopted:

(a) The sampling method for the test on engine 'A' was the so called 'Impinger Method'. A heated (130°C) sampling line of stainless steel tube was connected to an impinger where exhaust gas was bubbled through 25 ml. of a 3-methyl-2 benzo thiozalone hydrazone (MBTH)

solution. The exhaust was drawn through the impinger by a diaphragm pump. Flow rate was controlled by a needle valve and monitored with a rotameter. Sampling was done for 1 min. at 130 cc/min.

(b) The sampling technique adapted for the tests on engine 'B' was by the so-called 'Trichter' method adopted by Technische Hochschule, Aachen, West Germany (16). A 400 ml 'Trichter' flask containing 50 ml. of MBTH solution was evacuated to 100 mm of Hg. and connected to exhaust tapping. The exhaust gas surged into the flask for 15 seconds. Then the flask with the solution was allowed to stand for an hour for the reaction to be complete. After an hour, 10 ml of the above solution from the flask was taken in 50 ml flask. To this 1 ml. each of ferric chloride and sulfamic acid (of concentration 1.0 per cent and 1.6 per cent respectively) were added and the contents made up to 50 ml. with distilled water. After mixing and allowing 20 minutes for colour development, the absorbance was determined on the spectrophotometer at 628 nanometers (nm), with distilled water as reference medium. The aldehyde emission was calculated using a calibration curve.

RESULTS AND DISCUSSION

INFLUENCE OF ENGINE PARAMETERS ON ALDEHYDES - In all cases (excepting variable load tests) tests were conducted with no load to isolate the effects of other parameters which influenced aldehyde formation under load conditions. Results discussed are based on experiments conducted on engine 'A' using both gasoline and methanol.

Fuel Air Equivalence Ratio and Compression Ratio - Figure 1 shows the influence of equivalence ratio on aldehyde emission. The change in equivalence ratio essentially changes exhaust temperature and oxygen concentration both of which are important potentials for aldehyde reactions. Around stoichiometric air fuel ratio (0.0 in the present case) the high temperature produced minimum aldehydes and the level increased as the mixture was made richer or leaner. Recall that flame speed is maximum around stoichiometric ratio which improves combustion and reduces quenching effect. Compared with gasoline the level of

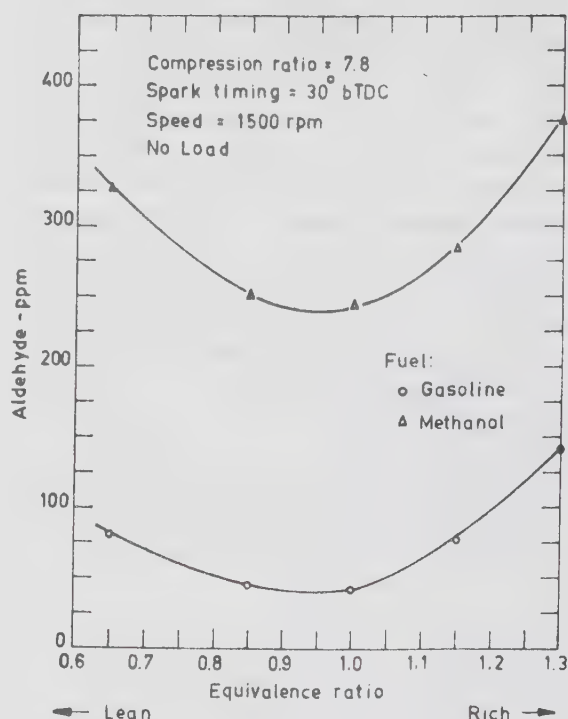


Fig.1 Effect of Equivalence ratio on Aldehyde emission

aldehydes increased by a factor of 4, depending on equivalence ratio. A similar trend for ethanol was reported in reference (4). See also the results shown on Fig.8, obtained on engine B, which was working on rated load. Here, the aldehyde profile is different in the rich region presumably under the concurrent effect of load.

Compression ratio effects are shown on Fig.2 (see also Fig.9) Aldehyde emissions were highest at 12 CR. Bernhardt (25) found that aldehyde emissions were highest at 12 CR and decreased with increasing CR above 12. According to Pischinger and Kramer (24) the influence of CR on aldehyde emission was not uniform. In the lean range, aldehydes always increased with increase of CR. CR effects on ethanol have been studied by Brinkman (4) from 7.5 to 18 CR. Throughout the range of the CR, aldehyde emissions increased.

The first cause for this increase in aldehydes is the reduced exhaust temperature consequent to the increased thermal efficiency compatible with high

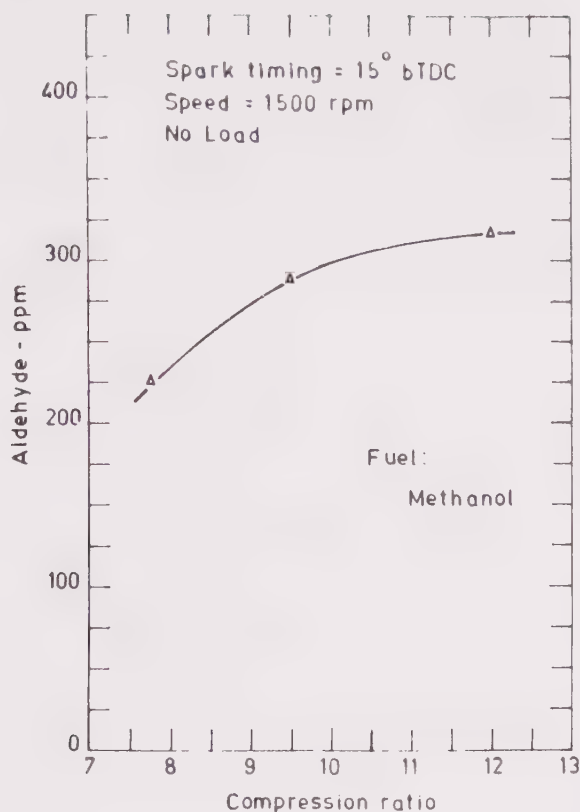


Fig. 2 Effect of compression ratio on Aldehyde emission

compression ratio. Partial oxidation of unburned fuel (UBF) takes place in the exhaust passageway instead of more complete oxidation encountered at higher temperature of low CR. This reasoning is supported by thermokinetic consideration of reference 13.

Discussion on the compression ratio effects on engine 'B' working under rated load is presented later with reference to results shown on Fig. 9.

Speed and load changes - With increase of speed, aldehyde emissions reduced for both methanol and gasoline (Fig. 3). The explanations for the drastic reduction are (a) increased flame speed, combustion temperature and better combustion resulting from higher speeds (18), (b) more effective eddy diffusion of UBF from quench layer to the bulk gas region, thus bypassing low temperature oxidation (12), (c) increased exhaust port turbulence at higher speeds promoting better mixing, (d) increased average exhaust temperature resulting from high speed.

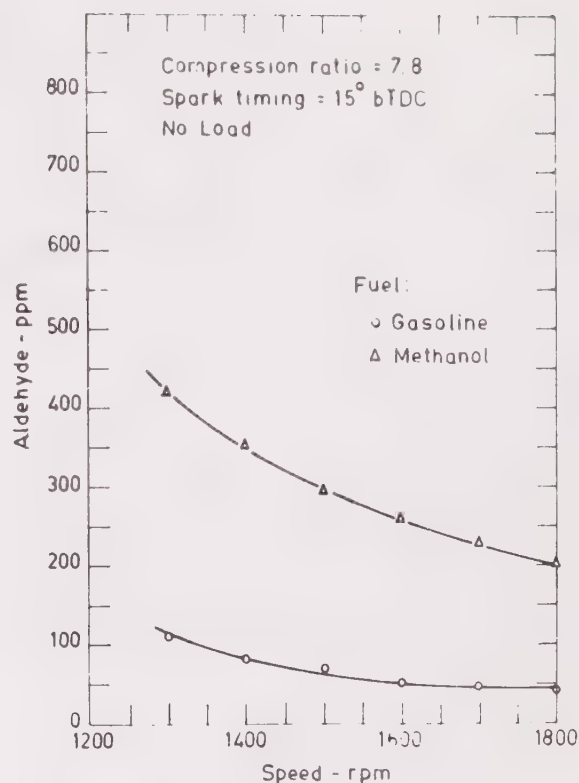


Fig. 3 Effect of speed on Aldehyde emission

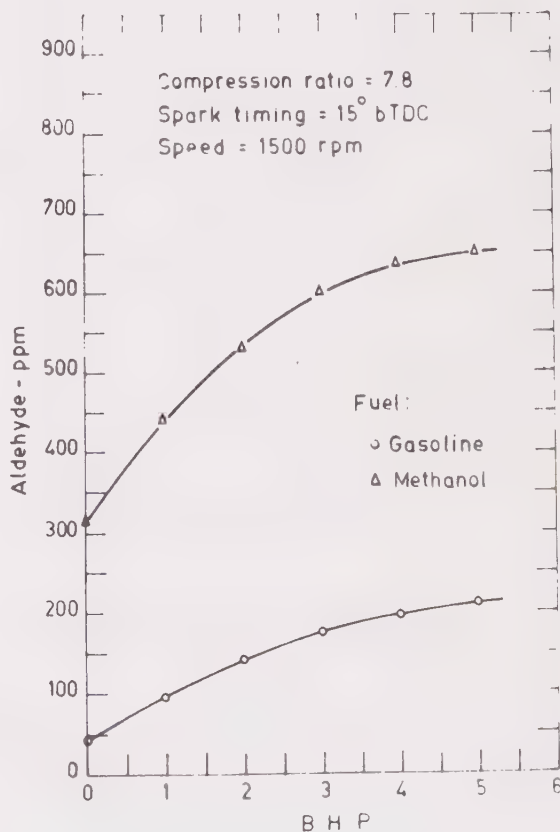


Fig. 4 Effect of load on Aldehyde emission

Of these factors, the authors are of the opinion that factor (b) is the most dominant.

Effect of load on exhaust aldehyde level is shown on Fig. 4. With both methanol and gasoline there was a marked increase of aldehyde emission as load increased. The reasons for this are (a) reduced residence time within the exhaust system at higher loads and (b) more mass of UBF in the quench zone accompanying higher load. Residence time is defined (26) as the ratio of volume of hot exhaust system (m^3) to volume flow of exhaust gases (m^3/min). If the load is doubled, volume flow is approximately doubled and residence time is reduced by 50 per cent. The increased mass of unburned fuel in the quench zone is, in the opinion of the authors, the more important cause, overriding other factors like increased temperature.

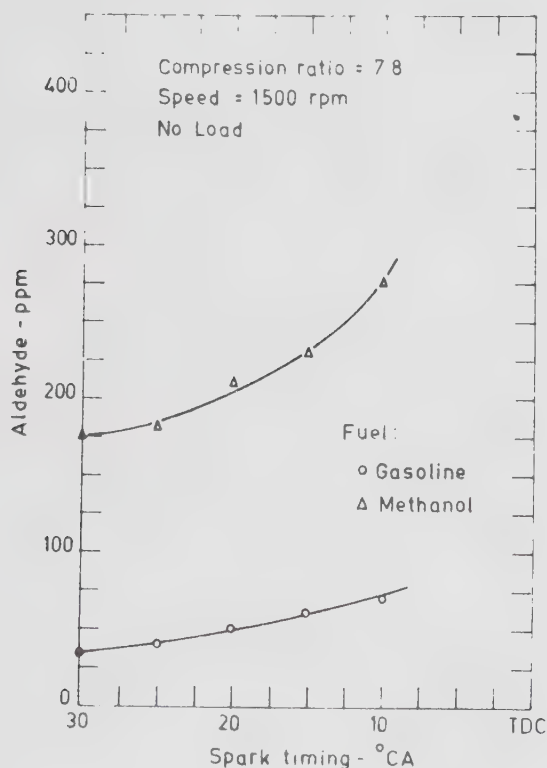


Fig. 5 Effect of Spark Timing on Aldehyde emission

Spark timing effects - Fig. 5 shows influence of spark setting on aldehyde emission. In the case of methanol, aldehyde level increases steeply as it is retarded from 30° BTDC to 10° BTDC. With gasoline there is no significant

change in aldehyde emission with spark timing. This finding agrees with the results of Fischinger and Kramer (24). With retarded spark less time is available for complete combustion. Owing to incomplete combustion the quench layer thickness may also increase contributing to higher aldehyde emissions. The difference between the aldehyde emission levels in Figs. 1 and 5 at 30° BTDC spark timing is due to the fact that the cooling water flow rates were different.

INFLUENCE OF FUEL TREATMENT -

Results discussed are based on experiments conducted on engine 'B' as specified in Table 2.

Effect of adding aniline - A thermodynamic analysis of the measured cylinder pressure traces at compression ratios of 7 and 11 was carried out through a computer program described in reference (17) and (18) and run on IBM system 370/155. Computed values of burned gas temperature and position of flame from the spark plug forward for methanol and 99 percent methanol with 1 per cent aniline are plotted in figures 6 and 7. Tables 3 and 4 give average flame speeds, peak combustion temperatures and history of aldehyde and NO_x emission with other important experimental results.

The most significant observation was the substantial reductions of aldehydes with the addition of 1 per cent by volume of aniline. The aldehyde concentration reduced by 10.18 per cent at a CR of 7 while this concentration reduced by 54.42 per cent at a CR of 11. The probable explanation for this drastic reduction is offered by the results of computed values and of burning rate and peak temperature presented in figures 6 and 7. Note that the peak temperature and flame velocity increased with addition of 1 per cent aniline which is a known ignition accelerator. With high temperature and burning rate, the mass in the quench envelope decreased along with reduced exposure time for the mass in quench envelope for in-cylinder aldehyde reactions. Further, aniline is known to reduce formaldehyde formation in the end gas and quench layer. According to Wihrow and Rass-Weiler (7) formaldehyde formation is suppressed

Table 3 - Computer Prediction of Flame Speed Peak Combustion Temperature, indicated thermal efficiency and indicated mean effective pressure at stoichiometric condition and MBT-Spark Timing

Fuel	CR	Average Flame speed- metre/ sec.	Peak combustion temperature °K	Indicated thermal efficiency %	Indicated mean effe- ctive pres- sure Kg/cm ²
1. 100% Methanol	7	13.76	2499.35	25.48	3.772
2. 1% Aniline + 99% Methanol	7	19.87	2542.00	29.01	4.773
3. 100% Methanol	11	16.76	2600.00	28.56	4.968
4. 1% Aniline + 99% Methanol	11	21.11	2643.61	30.15	5.087

C.R. = Compression Ratio

Table 4 - Experimental results of Aldehyde and NO_x emissions and exhaust temperatures at stoichiometric condition and MBT - Spark timing.

Fuel	C.R.	Aldehyde in PPM	NO _x in PPM	Exhaust temp. °C	Per centage reduction in aldehyde for aniline addi- tion	Percentage increase in NO _x for aniline addition
1. 100% Methanol	7	393	1025	552.0		
2. 1% Aniline + 99% Methanol	7	353	1097	592.0	10.18	7.02
3. 100% Methanol	11	757	1057	569.0		
4. 1% Aniline + 99% Methanol		345	1325	718.0	54.42	25.35

C.R. = Compression Ratio.

in the non-inflamed gases by the addition of aniline.

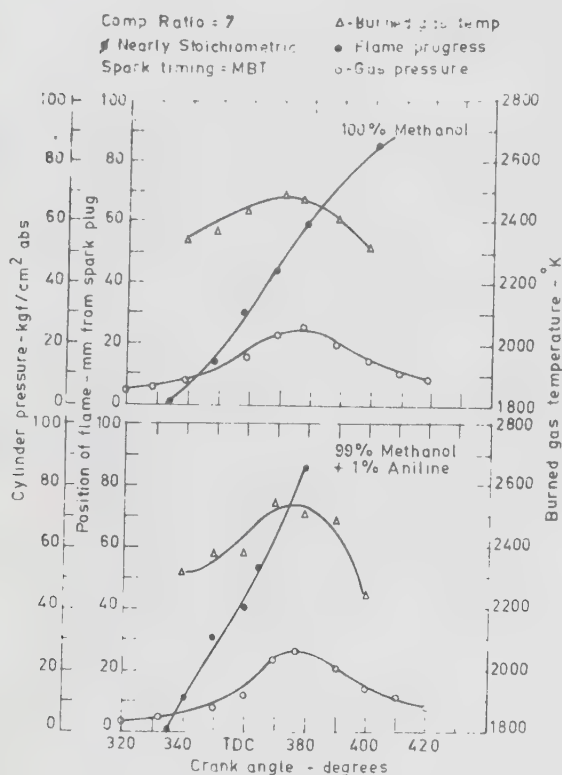


Fig. 6 Effect of Aniline addition

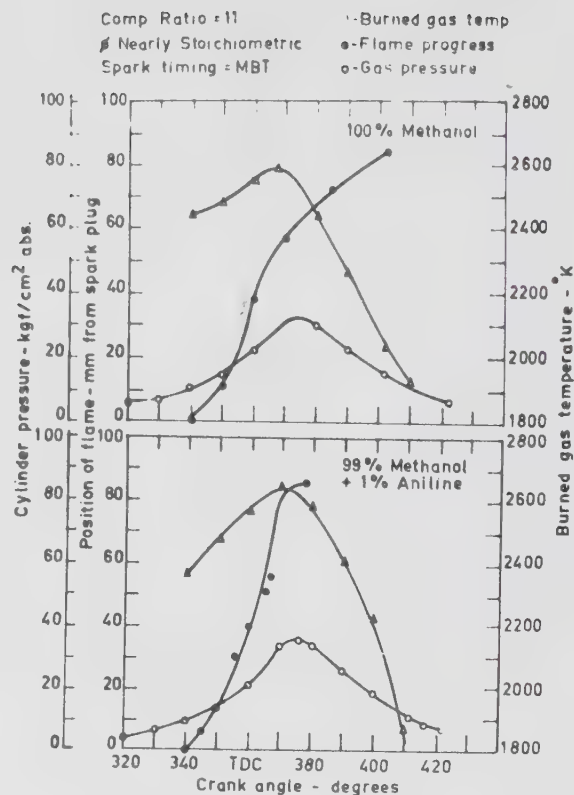


Fig. 7 Effect of Aniline addition

The effect of aniline additive at various equivalence ratios is shown on Fig.8. The first observation is that 0.75 per cent aniline is more effective in suppression of aldehydes than 1 per cent addition. Aniline belongs a class of additives which reduces ignition delay in small concentration and increases in large concentrations. The critical concentration was found to be around 0.8 by volume from engine tests (19). The second observation is that the exhaust UBF and aldehyde profiles are different. The exhaust UBF profile is the characteristic U shaped curve reaching a minimum at approximately $\phi = 0.83$, which closely agrees with the findings of Pefley, et al. (20) and Hilden et al. (21). The aldehyde profile both with aniline and without aniline is a steadily decreasing curve. As predicted by the computations of Brown and Pefley (11) excess oxygen and high exhaust temperature on the leanside have resulted in higher aldehydes concentration while the lack of oxygen and

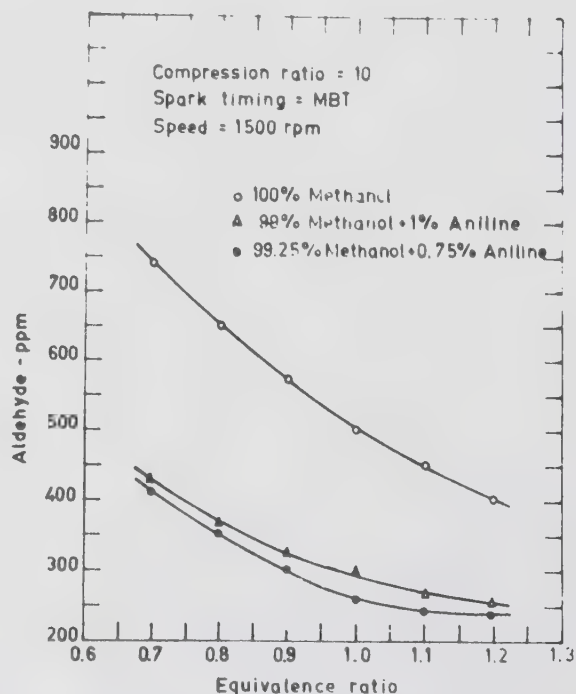


Fig. 8 Effect of Aniline on Aldehyde emission at various equivalence ratios.

lower exhaust temperature on the rich side have resulted in lower aldehydes.

The effects of compression ratio on aldehydes emission with neat methanol and methanol with aniline additive is presented in Fig.9. With neat methanol the aldehyde concentration increased from 390 to 760 ppm (93 per cent) with increase of CR from 8 to 12. Comparing this with the profile of UBF, there is an increase from 5700 ppm to 6700 ppm (18 per cent).

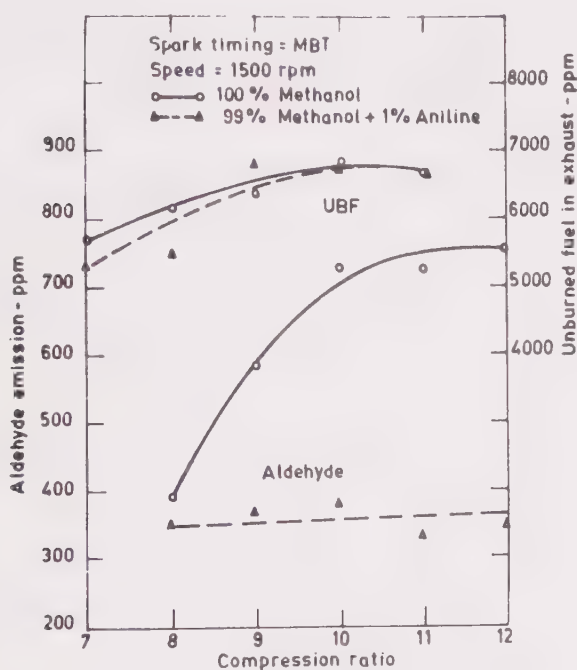


Fig.9 Effect of aniline addition on aldehyde and UBF at various compression ratios and at stoichiometric without preheat

Notice that in the addition of aniline, compression ratio has no significant effect on the aldehyde concentration which is maintained around 360 ppm. The UBF profile on the other hand follows the same course as UBF profile without aniline. This tends to support the view that bulk of aldehyde formation is confined to in-cylinder process and the action of aniline lies in suppressing the excessive formation of aldehydes in the cylinder. This view is quite possible since (a) Large quantities of liquid alcohol are inducted during the suction stroke on account of the higher stoichiometric fuel-air ratio and latent heat

of alcohols. (b) The crevices in the cylinder walls offer pockets for this liquid fuel to hide, as the fuel is spread over the cylinder walls during the compression and expansion strokes, thinning sufficiently to evaporate late in the expansion stroke and undergo slow oxidation reaction leading to aldehydes. (c) Mathematical flame quench modelling does not make necessary correction for this initial spread of methanol on the surface.

Kim et al.(10) support this view in respect of unevaporated fuel in the combustion chamber.

However, addition of aniline is not totally an unmixed blessing. Aniline ($C_6H_5NH_2$) is an aromatic compound with nitrogen bound in the molecule. It may be argued that the fuel bound nitrogen give rise to higher NO_x levels even though the quantity of NO_x on this account may be negligible. Moreover the overriding consideration is the higher peak combustion temperatures with aniline addition. The computed temperatures and measured NO_x emission (Tables 3 and 4) lend support to this argument. Aniline being an aromatic, may produce harmful emissions in exhaust if emitted unburnt.

Effect of adding water - Burned gas temperature computed from the pressure crank angle diagrams for 100 per cent methanol and 90 per cent methanol with 10 per cent (by volume) water blend, for CR of 9 and 11 respectively are plotted on Figs. 10 and 11. Tables 5 and 6 give the test conditions and the corresponding results of aldehyde and NO_x emissions.

The first observation is the reduction of peak combustion temperature at both the compression ratios with water addition. There is a consequent reduction of NO_x by 31.09 per cent at 9 CR and by 43.72 per cent at 11 CR. This reduction is in conformity with the computer predictions of Browning and Pefley (11). As a penalty the reduction of NO_x is accompanied by a reduction in power and thermal efficiency.

The second and more significant

Table 5 - Computer prediction of peak combustion temperature, indicated thermal efficiency and indicated mean effective pressure at stoichiometric condition and MBT - Spark Timing.

Fuel	C.R.	Peak Combustion Temperature °K	Indicated Thermal Efficiency %	Indicated Mean Effective Pressure Kg/Cm ²
1. 100% Methanol	9	2494.00	24.17	3.947
2. 10% Water + 90% Methanol	9	2360.00	20.40	3.477
3. 100% Methanol	11	2504.95	28.56	4.963
4. 10% Water + 90% Methanol	11	2395.00	25.00	4.523

C.R. = Compression Ratio

Table - 6 - Experimental Results of NO_x Aldehyde emissions and exhaust temperatures at stoichiometric condition and MBT - Spark Timing

Fuel	C.R.	Aldehyde in PPM	NO _x in PPM	Exhaust temp. °C	Percentage reduction in aldehyde for Water addition	Percentage reduction in NO _x for Water addition
1. 100% Methanol	9	729	1039	579		
2. 10% Water + 90% Methanol	9	583	716	540	20.02	31.09
3. 100% Methanol	11	757	1057	569		
4. 10% Water + 90% Methanol	11	426	810	550	43.72	43.72

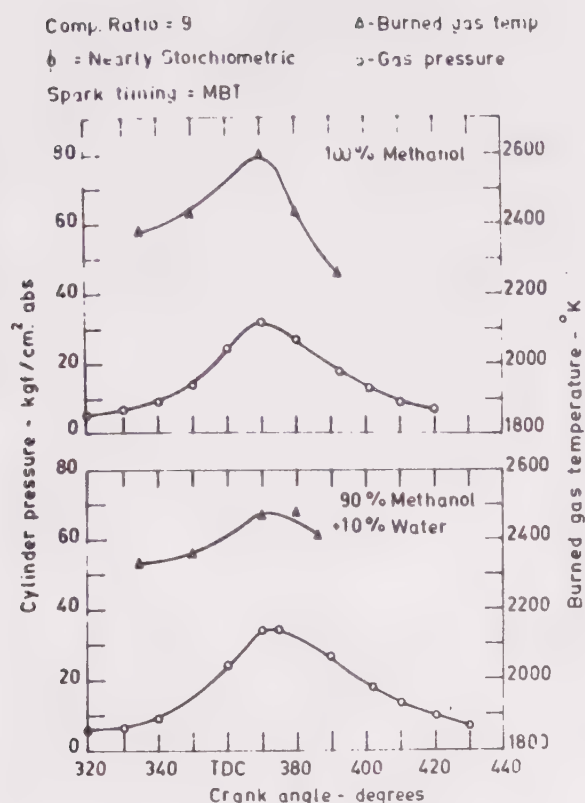


Fig. 10 Effect of Water Blend

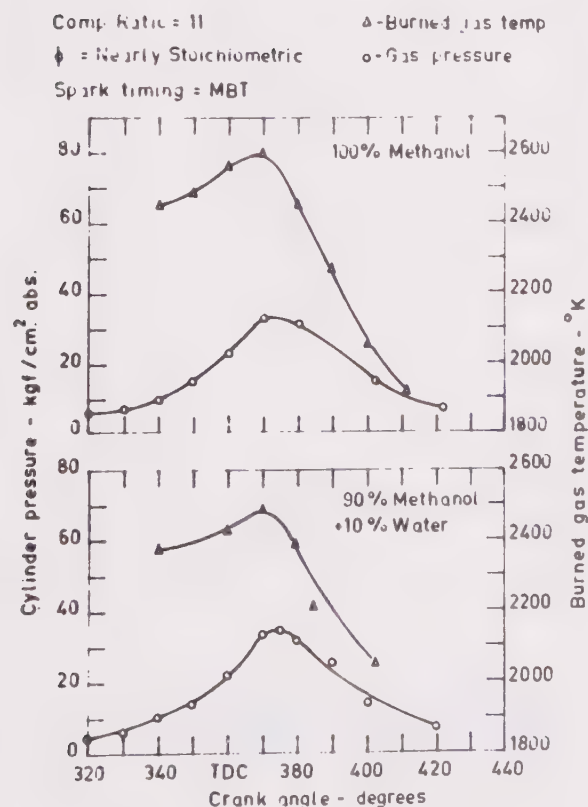


Fig. 11 Effect of Water Blend

observation is the reduction in the aldehyde emissions with 10 per cent (by volume) of water addition reduction of 20.02 per cent and 43.72 per cent at 9 and 11 CR's respectively. This reduction of aldehyde with water addition was also reported by Volkswagen researchers (23) who suggested water addition to methanol as one of the ways reducing aldehyde emissions. Their report was based on a trial of a 4-cylinder engine (full throttle and 2000 rpm). Addition of 5 per cent, 10 per cent, and 15 per cent water to methanol gave a progressive reduction of aldehyde emission as water content increased. Hydrocarbon emission was more or less steady upto 10 per cent of water but increased steeply by increasing water from 10 to 15 per cent. There was also progressive loss of power as water content increased. Quite contrary results were reported by Pischinger and Kramer (24) who worked on two different combustion chamber configurations one with a piston-bowl combustion chamber, 8 CR and MBT spark timing and the other with disc shaped chamber with flat piston, 8 CR, spark at 40° CA BTDC. In both engines, increasing water content in methanol produced increasing aldehyde emissions especially near the stoichiometric air fuel ratio. The aldehyde emission around stoichiometric ratio was found to be higher by a factor of 4 with 10 per cent water content as compared to straight methanol.

These conflicting results need explanation. Based on the analytical study of aldehyde formation, exhaust temperature has strong influence on aldehyde emissions. Brinkman (4) made the following linear regression of aldehyde emissions versus exhaust temperature, using the ethanol data on a CFR engine test.

Aldehyde emission $\mu\text{g/J} = 1.959 - 0.003035 \times t^{\circ}\text{C}$. The negative sign for the coefficient for exhaust temperature readily explains the results of Pischinger and Kramer (24). As water is blended in methanol, drop in temperature of exhaust results in increased aldehyde emission from the above equation. Besides exhaust temperature, oxygen concentration and exhaust system residence time are also equally strong potentials for formation of aldehydes in exhaust (13, 14).

On the contrary, it is very likely that addition of water to methanol may influence the thermokinetic climate prevailing within the engine cylinder unfavourable for aldehyde reactions - a plausible explanation upholding the in-cylinder formation theory of aldehydes. Recall that water addition reduces knocking tendency and drastically cuts down the aldehyde reactions (7) in the unburned fuel evaporating from the cylinder walls late in the expansion stroke. This seems to be the main cause for reduced aldehydes with water addition as found in the present investigation as well as in the findings of Volkswagen (23).

Finally, the influence of water on aldehyde emission does not seem to be uniform. The configuration of combustion chamber has an influence on aldehyde emission. According to reference 24, with a lower CR, the piston-bowl type and at higher CR the disc shaped type proved to be the more favourable. With 10 per cent water additives the influence of CR may be reversed (24). Air fuel ratio, compression ratio, spark timing and engine speed may all influence the formation either in-cylinder or in the exhaust passage way. But the evidence of both aniline addition and water addition emphasize the point that aldehyde formation in the in-cylinder process is considerable and cannot be neglected as compared with the aldehyde formed during the exhaust process.

CONTROL METHODS

ANILINE ADDITIVE - This is a significant method of reducing the aldehydes during the formation process itself. But this may give rise to undesirable aromatic compounds, in the exhaust if some aniline escapes unburnt. Unless further exhaust analysis assures that all aniline has reacted in the cylinder itself, the method cannot be recommended. Moreover, the NO_x in exhaust should not exceed beyond the prescribed standards.

WATER BLENDING - This is an effective method as it reduces both aldehydes and NO_x emissions. Further results on aldehyde reduction with varying quantities of water at different load and speeds on the engine 'A' are shown in Figs. 12 and 13.

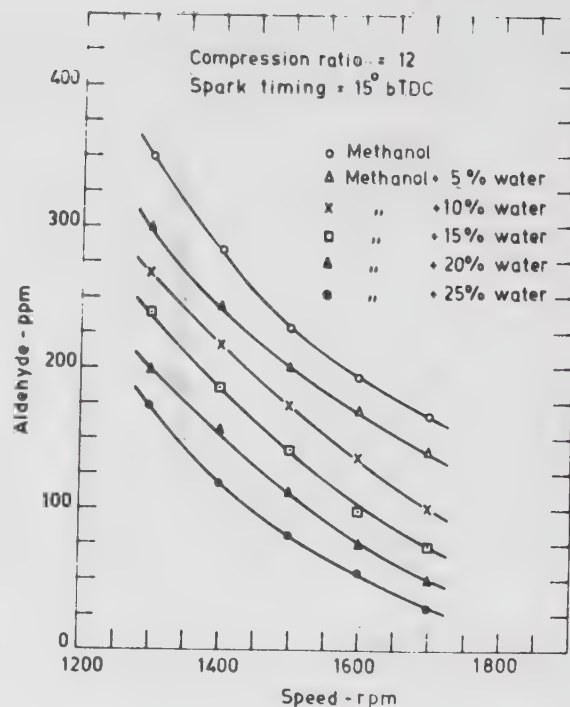


Fig. 12 Effect of blending Water with Methanol on Aldehyde emission

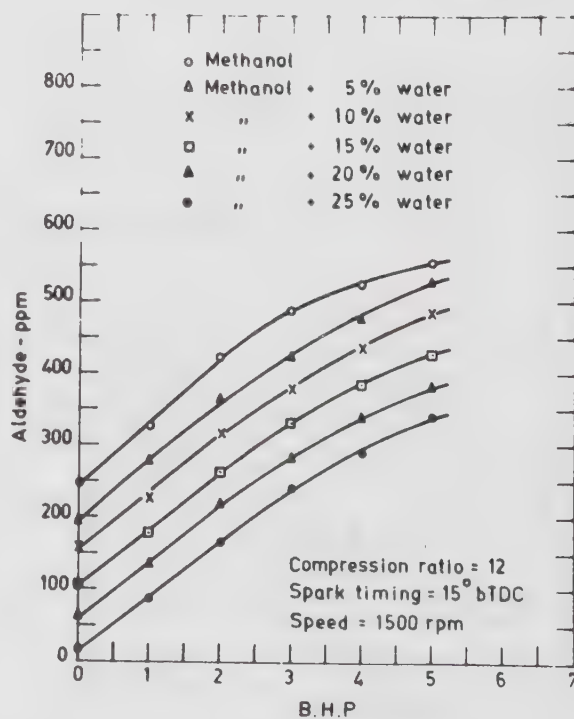


Fig. 13 Effect of blending Water with Methanol on Aldehyde emission

At each speed and load aldehyde emission reduced as the percentage of water in blend increased from 5 per cent to 25 per cent (by volume). The effect of increasing speed was to decrease the aldehydes for each blend. Octane rating improvement with water addition permitted operation at 12 CR without any knocking.

INLET AIR PRE-HEATING - Fig. 14 shows the extent of aldehyde reduction by preheating air to 60°C and 80°C. At maximum output there was a reduction of about 75 per cent and 125 per cent. The adverse effects on power and NO_x emission need to be investigated further before evaluating the effectiveness of this method.

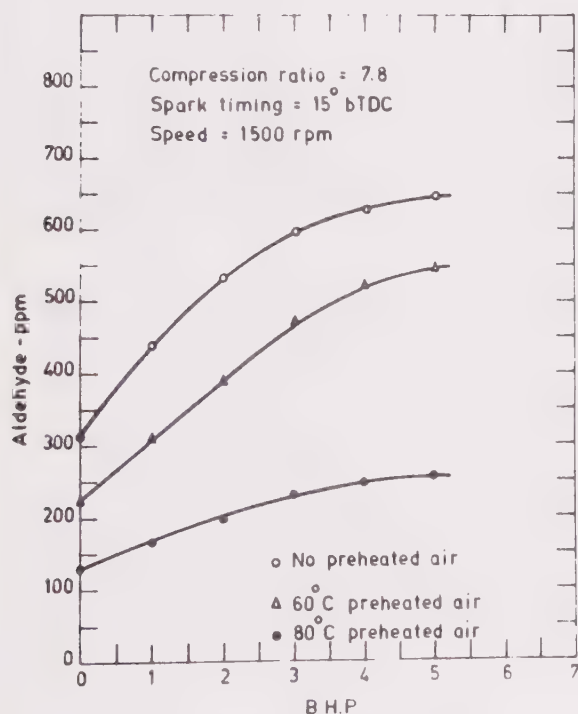


Fig. 14 Effect on preheating inlet air on aldehyde emission

SECONDARY AIR INJECTION IN EXHAUST LINE - Fig. 15 depicts a comparison of aldehyde levels without air injection and with secondary air injection into the exhaust line at rates of 0.05 m³/min. corresponding to 12 per cent and 28 per cent of primary air respectively. The percentage reduction in aldehydes for these two rates at the maximum power were 31 per cent and 42 per cent respectively.

REGULATION OF COOLING WATER - Effect of cooling water regulation on

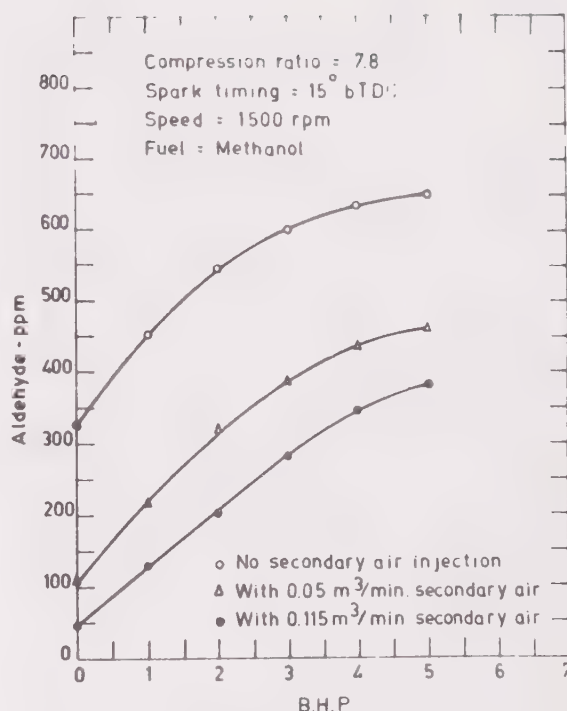


Fig. 15 Effect of secondary air injection into the exhaust line on Aldehyde emission

aldehyde emission fuel appears on Fig. 16. The emission level at two rates of cooling of 1.5 litres/min. with outlet temperature of 60°C and 2.5 litres/min. with outlet temperature of 50°C showed a reduction of about 25 per cent of aldehyde emission level.

AFTER TREATMENT OF EXHAUST -

Pischinger and Kramer (24) examined the influence of a platinum rhodium catalyst on the aldehyde emissions at various speeds and loads on a four cylinder Otto cycle engine.

Aldehyde emissions in methanol operations is reported to have been reduced without supply of secondary air by about 50 per cent in the fuel rich range and upto about 90 per cent in the lean range. This suggests further exploration of exhausts after treatment with indigenous oxidation catalysts.

NEW DEVELOPMENT - It is found that surface ignition engine reduces remarkably the aldehyde emission level by a factor of 4 to 6 when the engine is operated on ethanol and methanol as compared to an S.I. engine operating on alcohol. The description of the phenomenon and details of the combustion chamber was reported earlier (22). The authors of

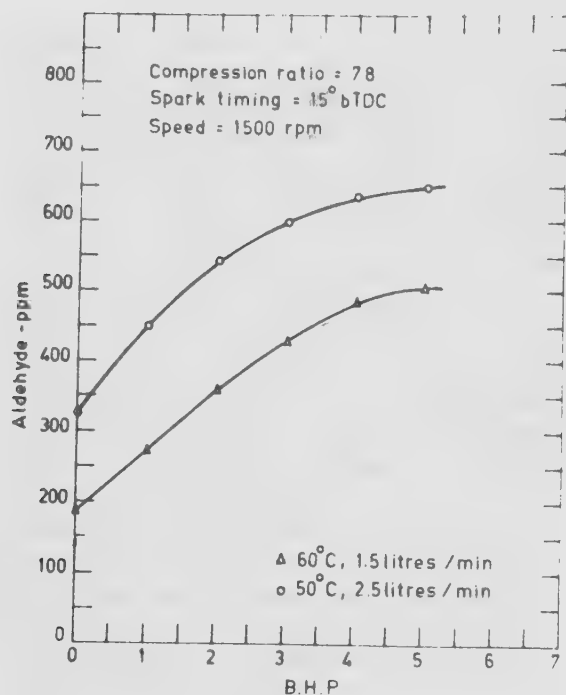


Fig. 16. Effect of reducing cooling water on aldehyde emission

this reference believe that the reason for this reduction is the absence of quench layer in this mode of combustion

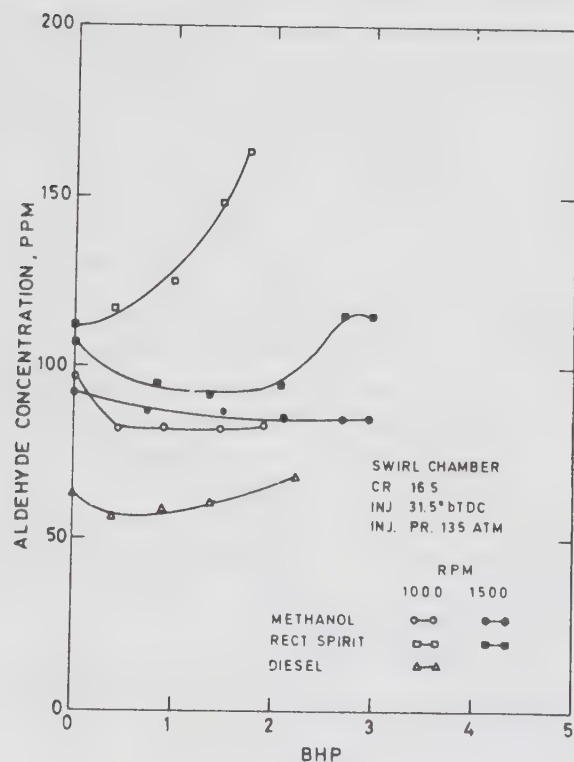
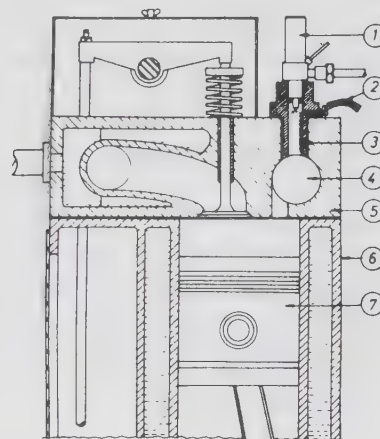


Fig. 17 Aldehyde Emissions in Surface Ignition Engine

A brief description of this engine is given in the Appendix. Fig. 17 shows the aldehyde emissions of this engine and Fig. 18 shows the details.



1 Pintle nozzle 2 Electrical Terminals
3 Asbestos surface with heating wires
4 Spherical-shaped swirl combustion chamber 5 Cylinder head 6 Cylinder
7 Piston

Fig. 18 High turbulence version of the surface ignition engine

CONCLUSIONS

(1) Available chemical kinetic models have been used to understand and explain the formaldehyde formation in the cylinder and exhaust system of a methanol operated S.I. engine. There seems to be little published data on reaction models predicting ethanol oxidation.

(2) With methanol operation, the influence of speed, load, spark timing, equivalence ratio and compression ratio on the exhaust formaldehyde emissions of a single cylinder engine has been established and compared with the contemporary work in literature. Corresponding results with ethanol found in literature are also presented. The trends and differences in results are explained in the light of the thermokinetic modelling.

(3) Increased engine operating temperatures or reduced cooling produce reductions in the aldehyde emissions.

(4) The conflicting results on the effect of water addition on the methanol

aldehyde emissions have given insights into the various competing and opposing influences on the formaldehyde formation. There is significant evidence on the formaldehyde formation in the in-cylinder slow oxidation process.

(5) The significant reduction of exhaust aldehydes resulting from addition of small quantities of aniline to methanol is explained with reference to enhanced burning rates from computer analysis of experimental data. These experiments also give strong evidence for sizable in-cylinder formation of aldehydes.

(6) Fuel additives like aniline as a control measure can only be recommended provided these additives do not produce other undesirable aromatics in the exhaust or increase the level of NO_x beyond regulated.

(7) Concept of surface ignition is indicated as a step towards new design trends for alcohol fuels with minimal aldehyde emissions.

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APPENDIX

High turbulence version of the Surface-ignition Engine

The high turbulence version of the Surface-ignition Engine incorporates a specially designed cylinder head. A mild-steel block containing the swirl chamber is bolted down into the cuboidal cavity (see Fig. 18). A holder, on the inside wall of which is mounted a surface igniter, is screwed into the mild-steel block, just over the swirl-chamber. The injector, screwed into the holder, sprays the alcohol so that it just grazes over the igniter and ignites. Thereafter combustion spreads rapidly due to the intense turbulence in swirl-chamber.

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THE DISTRIBUTION AND RETAILING OF FUEL METHANOL
IN THE SOUTH COAST AIR BASIN

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Prepared for:

The California Methanol Task Force

February 2, 1987

1. INTRODUCTION

The California Methanol Task Force* is holding a workshop to identify barriers to the large scale use of methanol as a transportation fuel and to solicit the advice of industry involved in the storage, distribution and retailing of motor vehicle fuels in Southern California. The workshop is the second in a series of government/industry workshops designed to identify and remove technical and economic barriers to the use of methanol in the transportation, industrial and utility sectors.

This document summarizes the results of a study completed by Energy and Environmental Analysis, Inc. (EEA), under joint funding from the U.S. Department of Energy and the U.S. Environmental Protection Agency. The report provides one perspective on the likely methods, equipment and costs associated with the use of fuel methanol in the South Coast Air Basin. This document is being distributed as background material for the workshop on methanol distribution cosponsored by the SCAQMD, ARB and CEC. The findings in this report will be a point of departure for much of the discussion at the workshop. The views expressed in this document are solely those of EEA and not necessarily those of the SCAQMD, ARB, CEC, EPA or DOE.

* The Task Force has been jointly established by the California Energy Commission, California Air Resources Board and the South Coast Air Quality Management District to promote the use of clean burning methanol as an air quality and energy security strategy.

2. SUMMARY

This document describes a methanol distribution system (equipment, logistics, and costs) for the South Coast Air Basin which will provide up to 250 million gallons per year of fuel methanol (an upper bound for methanol requirements in the Basin in the year 2000).

About 200,000 gallons per year of "near-neat" methanol currently is distributed for motor vehicle use in the South Coast Air Basin. The methanol arrives by rail tank car at an existing chemical storage terminal. The methanol is "splash" blended with 2 to 15% gasoline in dedicated tank trucks and is distributed through eleven specialized fleet service outlets in the area.

This existing distribution system can be expanded to a volume of 50 million gallons per year with the addition of more trucks and outlets for an estimated cost of \$1.2 million to \$9 million. Most of the cost is associated with modifications at the service stations. While demand is below 50 million gallons per year, too few service station owners may be willing to commit capital to a methanol service capability unless they can take a very substantial mark-up on the product or they are provided separate incentives by outside parties.

Expanding the distribution system from 50 million to 250 million gallons per year probably will require the modification of at least one existing petroleum product terminal to methanol handling. Changes could include dedicated handling and storage systems at a cost of \$7 million to \$8 million. Distribution to service stations would continue through the use of a dedicated truck fleet. The number of service stations would have to expand to 500 (ten percent of all outlets in the basin). At a volume of

250 million gallons per year, there would be both a sufficient number of outlets (from a customer perspective) and a sufficient sales volume (from the service station owners' perspective). The total capital cost of the terminal modifications, outlet changes and the tank truck fleet are on the order of \$5 million to \$27 million. The wide range in cost is due to uncertainties about the adaptability of existing petroleum product equipment to a limited methanol market.

EEA estimates that distribution activities (terminals, trucks, and service stations) will add 9¢ to 19¢ to the price of a gallon of fuel methanol.

3. EXISTING METHANOL DISTRIBUTION SYSTEM

3.1 METHANOL SUPPLIES

Methanol is not manufactured in California. Under contract to the California Energy Commission (CEC), Celanese Corporation supplies methanol from its Alberta, Canada plant for use in the state-sponsored demonstration methanol fueled fleets. Separately, Celanese provides methanol for direct purchases by Los Angeles County and Bank of America for use in their methanol vehicles.

One uncertainty regarding large scale use of methanol as a motor vehicle fuel is the product quality specifications which can or will be met by the product at the end of the distribution system (and placed in the vehicle fuel tank).

While chemical grade methanol is well over 99% pure, the quality requirements for fuel methanol are not as strict, and some contamination with water, organic sulfur and other hydrocarbons should be acceptable. Table 3-1 presents fuel specifications suggested by one methanol supplier and compares them to chemical grade specifications. Tolerable levels of contamination are not now known but these values are in the generally recognized range of acceptable levels.

3.2 TERMINAL AND BLENDING OPERATIONS

The methanol supplied by Celanese arrives in the South Coast Air Basin by rail tank car and is off loaded into a dedicated tank at GATX Terminaling Corporation's San Pedro Chemical Terminal. The tank has a cone roof with an internal floating roof. This combination protects against rain entry through the roof. A dedicated connection to the truck loading rack minimizes the chances of product contamination. The typical low volumes,

TABLE 3-1
FUEL METHANOL SPECIFICATIONS

<u>COMPONENT</u>	<u>CHEMICAL GRADE METHANOL</u>	<u>FUEL GRADE (DELIVERED)*</u>
METHANOL, % wt.	99.85	96.0
Water, % wt.	0.1	2.0
Hydrocarbons, % wt.**	-	2.0
Color, Apha	5	250
Trace Components (ppm)		
• chloride	-	<2
• sulfur	-	100
• iron	-	5
• sodium	-	5
• other metals	-	3

* As proposed by a methanol supplier

** In the gasoline/naphtha range

the segregation, and the cleanliness of the chemical terminal add 2-4¢/gal to the cost of methanol handling relative to gasoline.

The "near neat" methanol distributed by the California Energy Commission is 85 percent methanol blended with gasoline with a high aromatics content. Blending is done in the truck. The truck loads methanol at the chemical terminal and splash blends the 15 percent gasoline at a local petroleum products terminal.

3.3 DISTRIBUTION

The blended methanol fuel is delivered in a dedicated truck fleet. The cost of distribution by truck is on the order of 1.5¢ per gallon. No methanol is distributed by pipeline or through bulk plants. Even if one were not worried about contamination, the volume of methanol sales is too small to justify pipeline use.

3.4 METHANOL FUEL OUTLETS

There are eleven methanol fuel outlets in the South Coast Air Basin. Five outlets have been established by the California Energy Commission (CEC) at existing service stations and more are planned in the area. At these stations, the CEC installed all new tanks and pumps, designed specifically to be compatible with methanol. The equipment changes instituted by the CEC included the replacement of all pump seals with methanol compatible ones, the use of a chemical type hose, the replacement of the aluminum fuel screen with a stainless steel one, and installation of a flame arrestor on the system. Access to the CEC self-service pumps is provided using state-issued credit cards. Two fleet operators also have their own dedicated pumps. Bank of America has two outlets and the County of Los Angeles has four.

4. METHANOL DISTRIBUTION SYSTEM EQUIPMENT REQUIREMENTS

4.1 TERMINAL FACILITIES

As methanol use in the Basin increases, rail tank car deliveries probably will be augmented or replaced by vessel and barge deliveries. Any methanol terminaling facilities must be capable of receiving and handling the methanol without any substantial contamination with water, grit, or oil products (although some contamination with gasoline is acceptable). GATX's San Pedro Terminal is capable of handling methanol in a segregated clean system. Other private chemical terminals in the Los Angeles and Long Beach Harbor complex may have similar capabilities.

Use of a conventional petroleum product terminal carries with it some risk of product contamination. The reasons are several:

- Different products containing small amounts of H₂O are moved through common pipelines and manifolds.
- In some terminals, pipelines are flushed with water between changes in service.
- Water separates and segregates in low spots when the handling system is in petroleum product service. This water will be taken up when the system is switched to methanol service.
- Water heels (bottoms) are often kept in petroleum product tanks. A tank placed in methanol service must be drained and may require cleaning.
- The common double seal floating roof tanks, without a cover, have some risk of rain water leakage into the tank.

Many of these potential water sources can be eliminated by changing the operating practices at the product terminal. Particularly in the South Coast Air Basin, where rainfall is limited, it is likely that high volumes of methanol can be handled in conventional gasoline pipelines and tanks with acceptable levels of contamination. However, the necessary

safeguards, if that is not the case, should be identified and may be required while the market is in its infancy (as insurance against any possible problem while the fuel is being introduced).

One option would be to install separate lines and tankage for methanol handling at a petroleum product terminal. Another option would be to treat all other material entering the terminal with a dehydration unit.

If 100 percent methanol is not used and blending is required, meeting the prescribed "near neat" methanol specification can be accomplished in one of three ways:

- 1) Splash blending in the truck (the current practice): This approach is time consuming and may result in insufficient mixing in the truck. Product quality cannot be checked before shipping.
- 2) In-line blending as material is pumped to the truck: This minimizes the opportunities for the methanol to become contaminated but it does not allow product testing before shipping.
- 3) In-line blending to tankage: This is the common practice at refineries. Its key advantages are the ability to fine tune a blend to use the least cost components while meeting product specifications and testing and verifying product quality before shipment.

The second option is now favored for methanol product blending.

4.2 METHANOL DISTRIBUTION

As mentioned earlier, methanol is not handled in any product pipelines in the Basin. Common carrier lines are not expected to consider handling methanol until the volumes handled are very large (10-30 percent of the product traffic in the region), commingling of methanol product among shippers is acceptable, and the concerns about water contamination are resolved.

At the relatively small volumes of methanol traffic in the early years of its use, pipeline service offers no economic benefit in distributing

it within the Basin. Even at large volume the benefit of pipeline use (to supply in-land terminals) is on the order of 1/2¢/gal for the in-land markets.

Direct truck deliveries to service outlets is expected to be the predominant delivery method for methanol even when the sales volume is large. A fleet of dedicated tank trucks will minimize contamination but is marginally less efficient than the current practice of carrying multiple products in compartmentalized tanks, allowing the delivery of all products to a station in one truck.

4.3 METHANOL OUTLETS

The primary consideration in specifying methanol outlet equipment is materials compatibility. Underground storage tanks must be made of carbon steel or specially formulated fiberglass (conventional fiberglass can soften and deteriorate in the presence of methanol). State environmental laws require that service station tankage be monitored for leaks. As a result, some outlet tanks are being replaced with double wall tanks, and at other locations groundwater monitoring wells are being installed. Fiberglass tank manufacturers indicate that they are not getting many orders for methanol compatible fiberglass tanks, which presently cost \$1,000 to \$2,000 more due largely to the small volume of production of methanol compatible tanks. Also, current regulations do not require that tanks be both gasoline and methanol compatible. If an existing carbon steel tank is converted to methanol service, tank cleaning is recommended and a fuel filter is added to remove rust particles, which may be loosened from the tank walls by the methanol.

Service station pumps, pipes, and hoses need to be equipped to meet methanol's unique qualities, avoiding methanol contact with zinc or aluminum (corrosion) and certain plastics and rubbers (swelling or deterioration).

5. DISTRIBUTION SYSTEM EXPANSION SCENARIO

To identify prospective costs and logistical problems that might arise in the expansion of a fuel methanol supply system in the South Coast Air Basin, one technically feasible means of expanding the supply network has been developed. This expansion scenario is not intended to be a recommendation nor is it intended to imply that alternatives will be more expensive or less likely. In particular, this scenario focuses on a single terminaling operation serving the Basin. If methanol use grows substantially, one would expect several companies to enter the market and distribution would likely occur through several of the dozens of tank farms and terminals in the Basin.

The expansion scenario targets a sales volume of approximately 250 million gallons per year of methanol for vehicle use. This level of methanol use is considered an upper bound on what could be achieved by the year 2000 through an aggressive program of conversion to fuel methanol use. This level of demand can be achieved in several ways.

A sales volume of 250 million gallons per year is equivalent to methanol cars penetrating to 6% of the automobile population or a combination of 4% of the automobile population, 1 1/2% of the light and medium duty trucks and 50% of the bus fleet. Demand for methanol for use in gas turbines (such as those used in cogeneration projects) could represent forty million gallons per year of demand in the South Coast Air Basin by 2000 (based on ten facilities at 3 MW's capacity each firing methanol); and is equivalent to about a one percent penetration of the automobile population.

The distribution system expansion scenario has been specified in two phases. Phase I reflects an expansion of the current GATX chemical terminal operation. Phase II involves the installation of a large methanol distribution system at an existing petroleum product harbor terminal such as the GATX Carson Terminal. Table 5-1 outlines the modes of transport and storage in the current distribution system and the hypothesized Phase I and Phase II expansions.

In Phase I, the distribution system is expanded up to 50,000,000 gallons/year (versus less than 500,000 gallons per year at present). At this upper limit, loading of trucks would be carried out round-the-clock and at least 15 percent of the GATX terminal storage capacity is in methanol service. At the level of 50,000,000 gallons per year, the addition of methanol service at 100 to 250 service outlets is required. With 250 stations handling 50,000,000 gallons/year, methanol volumes at each station will be about half the volume now targeted per product for new stations. However, the volume is typical for the smaller volume existing outlets.

The Phase II system will handle 250,000,000 gallons per year of methanol. Any existing harbor product terminal can be modified to meet this requirement and a dedicated three bay truck loading rack could handle the associated round-the-clock truck loading. With the number of service outlets expanded to about ten percent of all outlets, methanol would be a high volume product at those 500 outlets, at or above the target throughput rates for a single product at a new service station. If the volume of methanol traffic in the South Coast Air Basin were to exceed 250,000,000 gallons/year, the system layout and cost per gallon are not expected to change dramatically from those described for Phase II.

TABLE 5-1
PROSPECTIVE EXPANSION OF METHANOL DISTRIBUTION
SYSTEM IN THE SOUTH COAST BASIN

Current System

Volumes:	Less than 500,000 gal/yr of methanol.
Terminal Receipts:	Rail tank cars or tank trucks.
Terminal:	GATX chemicals terminal with a dedicated tank and truck rack.
Blending: (if necessary)	Splash blended in the tank truck; truck makes a separate stop at a gasoline terminal.
Distribution:	Trucked directly to the outlets.
Outlets:	State leased space at service stations (5); privately owned fleet fuel stations (6).

Phase I Expansions (Maximum Use of Chemical Type Terminal)

Volumes:	500,000 to 50,000,000 gal/yr of methanol.
Terminal Receipts:	Chemical vessel, chemical barge or rail tank cars.
Terminal:	GATX or similar chemical terminal operation with dedicated lines from wharf to tank to trucks. Move to round-the-clock operation to meet increased volumes. Utilizes at least 15 percent of GATX chemical tankage and two of seven truck racks.
Blending: (if necessary)	Splash blended in the tank truck; truck makes a separate stop at a gasoline terminal.
Distribution:	Five dedicated trucks (tractor with two tank trailers) deliver directly to the outlets.
Outlets:	100 to 250 new outlets (at existing stations) -- represent two to five percent of all basin outlets.

TABLE 5-1 (cont'd)
 PROSPECTIVE EXPANSION OF METHANOL DISTRIBUTION
 SYSTEM IN THE SOUTH COAST BASIN

Phase II Expansion (Development of a Dedicated Methanol Terminal)

Volumes:	50,000,000 to 250,000,000 gal/yr of methanol.
Terminal Receipts:	Vessel, barge and railcar.
Terminal:	Expansion or modification of an existing L.A. harbor petroleum products terminal. Dedicated internal floating roof tanks and truck loading rack (three bays). Wharf to terminal pipeline may or may not be dedicated.
Blending: (if necessary)	Gasoline and methanol blended in-line during truck loading.
Distribution:	Dedicated truck fleet, delivers directly to outlets. No use of in-land terminals or common carrier pipelines.
Outlets:	Total of 500 outlets (methanol as a high volume product at ten percent of all basin outlets).

6. COST OF THE METHANOL DISTRIBUTION SYSTEM

Based upon the supply and distribution system expansion scenario in the previous section, EEA developed estimates of the capital investment and likely operating costs for both phases.

6.1 PHASE I CAPITAL COSTS

Table 6-1 presents EEA's estimate of the capital costs of creating a methanol distribution system handling 50,000,000 gallons/year. No additional capital investments are required for terminal operations. The GATX chemical terminal can receive by vessel, barge, or rail car these volumes, store the methanol in tankage with internal floating roofs and load trucks without any hardware changes. The volume will call for a switch to round-the-clock operations, however.

In distribution this volume of methanol can be handled easily by a dedicated fleet of five trucks (each delivering three to four truckloads per day). A single truck tractor with two tank trailers costs about \$140,000.

The largest potential cost will occur at the service outlets. A range of costs has been developed, keyed to the availability and suitability of in-place storage tanks. If an existing underground tank is suitable, the costs of cleaning it and modifying the pump are on the order of two thousand dollars. This lower cost case is credible under some circumstances because:

- Most stations will be phasing out leaded gasoline and will have a pump and tank available for an alternate product (medium octane unleaded is probably preferred).
- Methanol is a high octane product which logically could displace premium unleaded from its slot at a station.

TABLE 6-1
CAPITAL COSTS, METHANOL DISTRIBUTION - PHASE I EXPANSION
(at 50,000,000 gal/yr)

1. Terminal and Blending Operations

Existing chemical terminal facilities are adequate; no capital investment required.

2. Truck Fleet

Five trucks with tank trailers (8,500 gallon capacity)

5 x \$140,000 = \$700,000

3. Service Station Modifications

Low Cost Case: Uses existing tankage and pump. Costs are due to tank cleaning, new fuel screen, new fuel filter, and replacing seals and pump hose.

\$2,000 per station x 250 stations = \$500,000

High Cost Case: New double wall storage tank, new pipe to pump, and one new pump (methanol compatible)

\$33,000 x 250 stations = \$8.25 million

4. Total Capital Cost

Total capital cost = \$1.2 million to \$9 million

- In Phase I and Phase II, only five and ten percent of all service stations, respectively, are expected to handle methanol.

To present the full range of service station costs, EEA has estimated the cost of a new underground storage tank, pipe and a service pump. In EEA's review of the cost of a new tank, piping and pump resulted in a cost of about \$33,000 per outlet. At \$33,000 per station, this accounts for over \$8 million of potential costs related to the Phase I expansion.

The total estimated capital cost of Phase I is \$1.2 to \$9 million.

6.2 PHASE II CAPITAL COSTS

Capital costs for Phase II as presented in Table 6-2 include a substantial investment in terminal equipment. A high and low cost case were developed to reflect outstanding uncertainties. For the low cost case, terminal investments were limited to installing a new truck loading rack and an in-line blender. The low cost case assumes the use of existing tankage and harbor facilities. This case is credible (but not assured) for several reasons:

- The GATX Carson Terminal has sufficient tank capacity with internal floating roofs to handle the target 250,000,000 gallons/year. Currently these tanks are used by GATX clients and might be made available for methanol service in the future.
- While gasoline is comingled in most large product marine terminals, even "neat" fuel methanol can tolerate some gasoline addition (up to several percent) and can tolerate up to one percent water.
- There is generally an excess of product storage capacity in the petroleum industry due to recent efforts in inventory control.

However, the costs of all new tankage and a five mile dedicated pipeline to the harbor has been included in the higher cost case.

TABLE 6-2

CAPITAL COSTS, METHANOL DISTRIBUTION - PHASE II EXPANSION*
(at 250 million gal/yr)

1. Terminal and Blending Operations

Low Cost Case: Use existing wharf facilities (comingling with gasoline acceptable) and existing tankage. Install new truck rack and in-line blender.

Truck loading rack (three bays) - \$900,000
In-line blender - \$150,000
Total - \$1,050,000

High Cost Case: New dedicated pipeline from wharf to terminal, 500,000 bbls of new tankage with internal floating roofs, in-line blender, pumps and new three bay truck rack.

Pipeline to wharf - \$1.2 million
500,000 bbls storage - \$5.0 million
In-line blender and pumps - \$0.2 million
Truck rack - \$0.9 million
Total - \$7.3 million

2. Truck Fleet: Trucks with tank trailers - 22 x \$140,000 - \$3,080,000

3. Service Station Modifications

Low Cost Case: Using existing tankage.

500 stations x \$2,000 - \$1 million

High Cost Case: New tankage and two new pumps

500 stations x \$36,000 - \$18 million

4. Total Capital Cost - \$5.13 million to \$26.88 million

*Includes Phase I components.

For the Phase II expansion, the costs include 22 tank trucks and twice as many service outlets as specified in Phase I (each one handling more volume). The cost per station is \$3,000 greater to reflect the installation of a second pump.

The total capital cost associated with completing Phase II is between \$5 million and \$27 million.

6.3 IMPACT ON PRODUCT COST

The price of methanol at the retail outlet is expected to reflect the operating and capital costs of terminal, truck and service station operations. The current mark-up on gasoline, excluding taxes, is approximately 1¢/gallon for terminaling, 1¢/gallon for truck delivery and 7¢/gallon for service station operations. Methanol distribution costs are likely to be higher early in the market development due to capital investment requirements and smaller sales volumes.

Several independent retailers have indicated that generally they will not make a capital investment unless it can be recovered in three to five years. Table 6-3 shows the cost per gallon of methanol to recover the capital investments associated with both Phases over 5 years. Total cost for Phase I is 0.8¢ to 3.9¢ per gallon. For Phase II the total cost per gallon drops to between 0.4¢ and 2.2¢ per gallon. These figures are based upon steady sales volumes of 50 million and 250 million gallons per year, for each phase respectively.

Capital cost recovery mark-up during the transition from current sales of less than 500 thousand gallons/year to the target Phase I volume of 50 million gallons/year could be substantially higher. However, there are no real economies of scale in the terminal and trucking operations specified for Phase I (the terminal incurred no capital costs and the five truck fleet could be brought into service gradually). Only the

TABLE 6-4
METHANOL SUPPLY AND DISTRIBUTION ESTIMATED COSTS*

Phase I (50 million gallons/year)

Terminaling	3-5¢/gal
Trucking	1-2¢/gal
Service Station	<u>10-12¢/gal</u>
Total	14-19¢/gal

Phase II (50 million-250 million gal/yr)

Terminaling	1-2¢/gal
Trucking	1-1.5¢/gal
Service Station	<u>7-9¢/gal</u>
Total	9-12.5¢/gal

* Excluding Federal, State and Local taxes. Based on the current mark-up for gasoline and estimates of costs to cover capital investments.

service station costs could be substantially higher in the early part of the transition to vehicle methanol use. EEA estimates that methanol sales could progress as follows if an aggressive fleet conversion and new car introduction program were initiated:

Year 1	10 million gallons/year
Year 2	20 million gallons/year
Year 3	40 million gallons/year
Year 4	60 million gallons/year
Year 5	80 million gallons/year

If 250 service stations (5% of all outlets) were required in the South Coast Air Basin to supply the introduction of methanol vehicles and each station owner sought to recover a \$33,000 investment during that five year period, the capital recovery mark-up alone would be:

$$\frac{250 \text{ stations} \times \$33,000}{210 \text{ million gallons}} = 3.9¢/\text{gallon}$$

This figure is reasonably close to the higher steady state figure for Phase I in Table 6-3. This estimate is very sensitive to the acceptable time period for capital investment recovery. If a three year capital recovery period is applied, this figure rises to:

$$\frac{250 \text{ stations} \times \$33,000}{70 \text{ million gallons}} = 12¢/\text{gallon}$$

Table 6-4 provides an estimate of the total cost to be incurred in distributing methanol under Phase I and Phase II. Terminaling, distribution and retail services for Phase I are estimated at 14-19¢/gallon not including taxes. In Phase II, per gallon costs are expected to drop to between 9 and 13.5¢/gallon, excluding taxes. These cost estimates combine qualitatively the current costs of petroleum and chemical product distribution and some premium to recover the added capital requirements for methanol distribution over a five year period. These costs may be low if current rates do not include any significant capital recovery and if the methanol equipment purchaser requires a very quick payback. Alternatively, these costs may be too high (on the "per gallon" basis) because methanol sales volumes eventually will be higher than gasoline volumes due to energy intensity differences.

TABLE 6-3
IMPACT OF DISTRIBUTION SYSTEM CAPITAL
IMPROVEMENTS ON THE COST OF METHANOL

Phase I: Capital costs recovered over 5 years at an annual sales rate of 50 million gallons/year

Terminal Costs	-	0¢/gallon
Truck Costs	-	0.3¢/gallon
Station Costs	-	<u>0.5-3.6¢/gallon</u>
Total	-	0.8-3.9¢/gallon

Phase II: Capital costs recovered over 5 years at an annual sales rate of 250 million gallons/year

Terminal Costs	-	.08-0.6¢/gallon
Truck Costs	-	.25¢/gallon
Station Costs	-	<u>.08-1.4¢/gallon</u>
Total	-	0.4-2.2¢/gallon

7. OUTSTANDING UNCERTAINTIES AND CONCERNS

While this report prescribes a methanol fuel distribution transition for the South Coast Air Basin from less than 500,000 gallons/year to over 250 million gallons/year, several large uncertainties exist which will influence the ultimate creation, costs, and acceptability of the distribution system. Key concerns identified in the conduct of this study are outlined below:

1. Standardizing fuel methanol specifications: A variety of neat and "near neat" methanol blends have been tested and demonstrated. However, a uniform specification has not been adopted.
2. Applicable environmental, health, and safety requirements. While methanol is currently covered by environmental regulations, it has yet to be addressed in the volumes and public proximity envisioned in this scenario. An early indication of how fuel methanol will be regulated will improve the confidence and commitment of private parties to the implementation of methanol distribution systems.
3. Establishing the initial outlets: Initially, when demand is well below the 50,000,000 gallon/year target, service station owners may be unwilling to commit to methanol unless they take a substantial mark-up (as is done with low volume sales of propane) or they are provided separate economic benefits by outside parties. The necessary incentives do not now exist.
4. Adaptability of existing equipment: The question of water take-up when methanol is placed in existing gasoline pipelines and tanks may be resolved only after additional experience is gained handling methanol as a fuel. If the existing petroleum product systems are dry enough and clean enough, substantial cost savings will occur. For underground service station storage, a "window of opportunity" exists to make tanks methanol compatible. Specifically, some tanks are being replaced over the next couple of years to meet environmental regulations. For modest additional costs, methanol compatible tanks can be specified. Retrofitting methanol compatible tankage is much more costly. The incentives or requirements for methanol compatibility in new tankage are not clear.

5. Financial impact on independent service station owners: The cost of making a service station methanol compatible can be a significant financial burden to small business, station owners. Their ability to absorb these costs and remain financially viable is uncertain.
6. Minimum number of outlets. While this report assumes that 500 service stations out of 5000 in the basin will provide adequate access to a fuel methanol supply, the number needed to insure public acceptance and commitment to this new fuel has not been established.

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